

Subject:

Respiratory

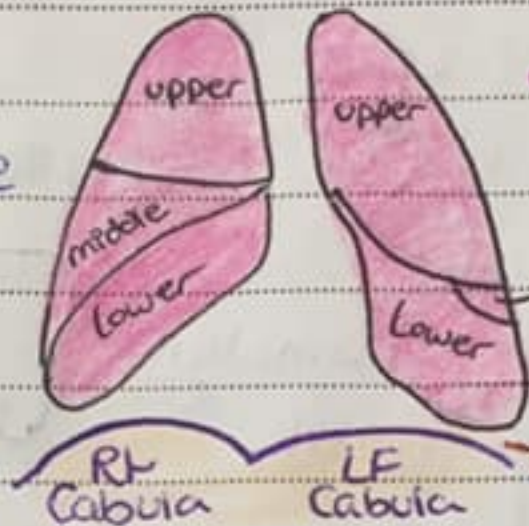


Introduction

in examination

- From front &

mainly Upper lobe
"up to 4th rib"



- From Axilla

middle lobe "Rt side"
lingula "LF side"

- From back &

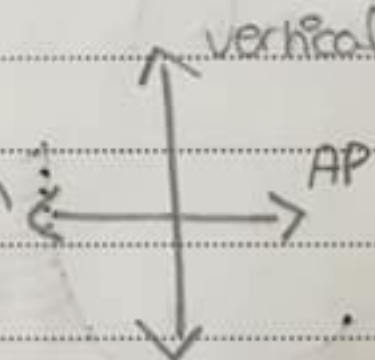
mainly Lower lobe

Diaphragm = main ms of Resp

Intercostal ms

→ accessory ms of resp "under stress"

→ ↑ Diameter of Chest AP



Supplied by Phrenic N

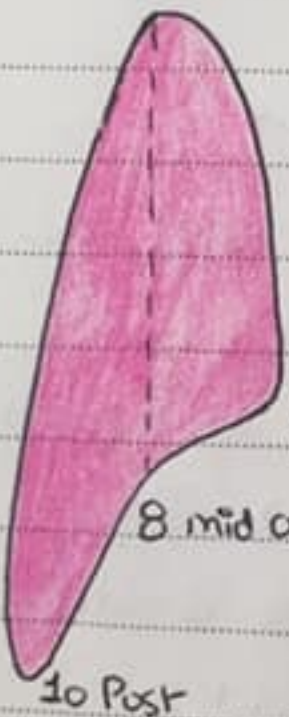
↓ contraction

↓ Down

↑ Chest cage vertically

→ ↓ pressure in Chest

(Inspiration)



- Lat view of Rt lung

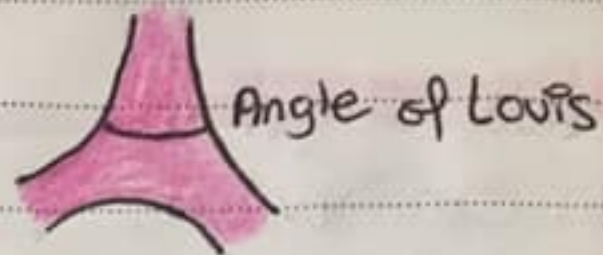
imp to know normal surface anatomy to detect certain type of Pathology

ex: Hyperinflated Chest (OAD) Collapse

VISIT...

LANZAROTE
Caliente.COM

- Airway $\Rightarrow$ Upper - lower
- Trachea Normally bifurcation



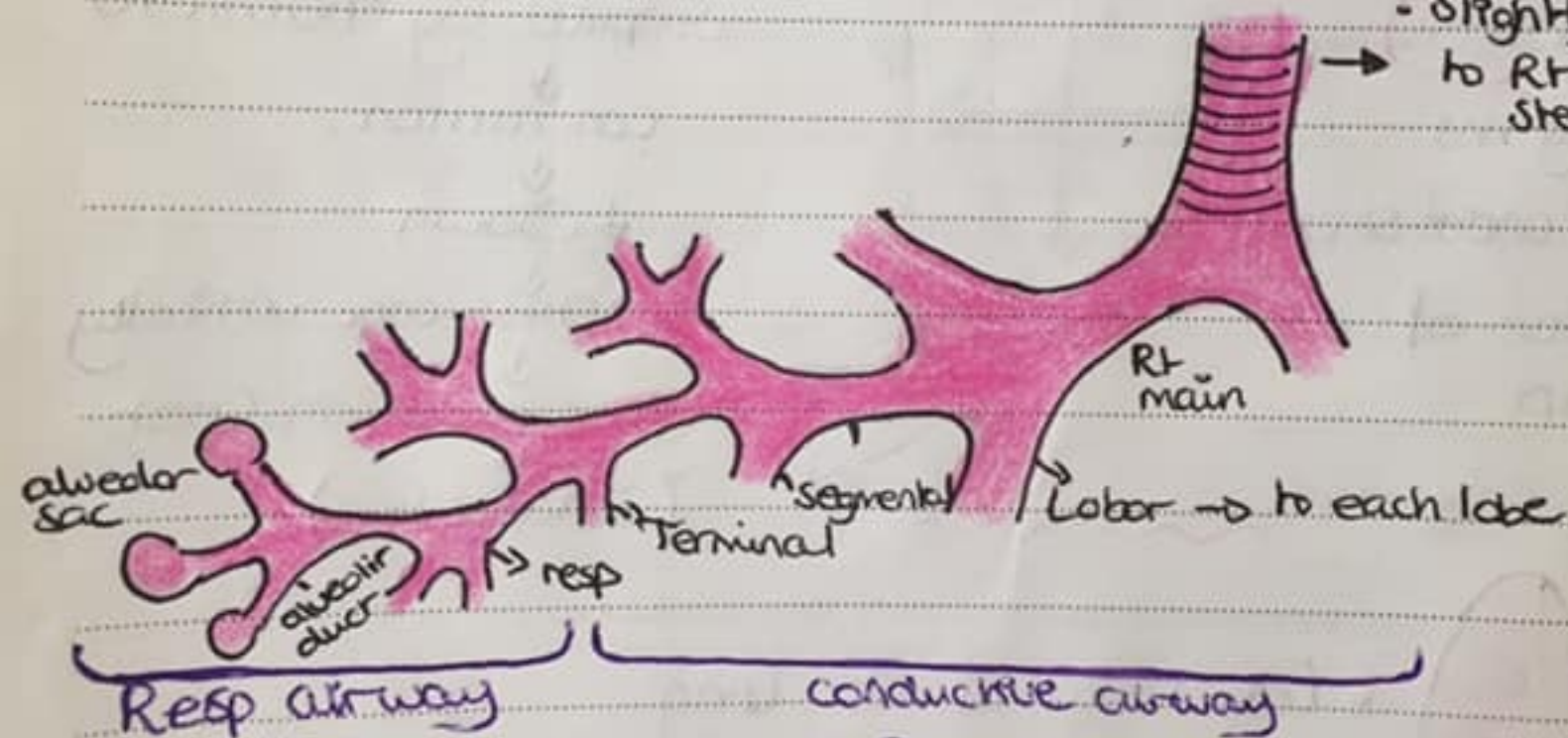
Ant $\Rightarrow$ Angle of Louis

Post $\Rightarrow$ at level of T₄ btw medial angle of scapula

- * So branchial breathing normally auscultated br-lat Para sternal at level of angle of Louis or At level of T₄ btw Scapula!



- Central Above
- Slightly Deviated to Rt behind Sternum



Functional unit of lung

"Gas exchange"

Physiological dead space from nose to terminal bronchiole

- Conduction of air to respiratory
- humidification $\rightarrow$ if Dry
- Filtration "by cilia & mucus $\rightarrow$ if Dust
- warming $\rightarrow$ if Cold

Histo

mucosa

Upper airway

"Pseudo-stratified Columnar
Ciliated epithelium with Goblet-cell"



for Protection from dust-etc

Goblet secret mucus &

Cilia move it up & Down

Lower airway

Single Columnar Not
ciliated epithelium with
less goblet cell!



for gas exchange

Note Kartagener Syndrome = immotile cilia syndrome

b/c Cilia not moving → mucus Plug → good media for

bact over growth → recurrent Chest infection

→ destruction of airway "Bronchiectasis"

Note Goblet cell b/c in upper > lower

in upper airway infection "Trachitis-bronchitis" →

irritation of goblet cell → Productive Cough

but in Lower airway infection "Pneumonia" → Dry Cough

* if Productive → from Tx affecting upper

or infection started upper then lower!

Submucosa

Upper airway

Lower airway

Blood Supply from
"Systemic Circulation"
= Bronchial a from Aorta

Blood Supply from
Pulmonary artery
from Rt $\heartsuit$

Note

b/c Systemic Pressure 120

Pulm Pressure 25 "at rest" / 30 "at exercise"

So Hemoptysis $\rightarrow$ massive "more than 0.5L" in upper
airway disease ex: Bronchiectasis
Bronchogenic Carcinoma
TB
 $\rightarrow$ minimal in lower airway ex: Pneumonia
Pulm edema

muscolasa

ms thickness in upper > lower

so in bronchospasm $\rightarrow$ b/c different ms size $\rightarrow$ different
in Power of Contraction $\rightarrow$ different Sound "Raché"

"Polyphonic = Systemic problem ex: Asthma

but if spasm uni-lat at one area $\rightarrow$ monophonic

DD Foreign Body
LoN or mass] Partial Obstruction



ms receptor

Sympathetic

Para-sympathetic

B₂ Adrenergic

Cholinergic 'muscarinic'

(Bronchodilation)

(Bronchospasm
+ goblet secretion)

Histamine receptor

Leukotrien receptor

Note

Tx of OAD

- B₂ Agonist (SABA-LABA)
- muscarinic Antagonist (SAMA-LAMA)
- Histamine Antagonist
- Leukotriene Antagonist

upper airway

Stridor thick ms

massive hemophysis

Productive Cough

less hypoxia

lower airway

wheezes thin ms

less hemophysis

Dry Cough

more hypoxia "b/c gas exchange"

Physiology

Inspiration

air from atmosphere
to alveoli "ventilation"

"Actively"



Contraction of Diaphragm
by impulse from Phrenic N



-ve Pressure in chest



air rushes in

Expiration

air from alveoli out
to Atmosphere

"Passively"



relaxation of Diaphragm
back to normal position



+ve Pressure



air goes out

- Cortex → voluntary signals to Diaphragm
 - Brain Stem → regulation of breathing
- So during night Cortex shut down, breathing is controlled by brain stem

★ Expiration by recoil of elastic fibers "Passively"

So Pt with elastic problem "emphysema" → can't
do good expiration → air-traps in → ↑ alveoli pressure
→ can't get air in → Dyspnea

* Centers of respiration in brainstem & medulla

"ventral & Dorsal Part"

have receptors for $(\text{CO}_2 + \text{H}^+)$ $\rightarrow$ Stim

by $\downarrow \text{pH}$ " $\uparrow \text{H}^+$ " or $\uparrow \text{CO}_2$

normal $\text{PaCO}_2 = 35 - 45 \text{ mmHg}$

if Pt hypercapnia $\rightarrow$ Stim respiratory center $\rightarrow$

$\uparrow$ rate & Depth $\rightarrow \text{CO}_2$ wash

Hypercapnia $\rightarrow$ acidify in blood b/c $\uparrow (\text{H}_3\text{CO}_2)$



Prolonged hypercapnia "as in COPD Pt" $\rightarrow \text{CO}_2 \uparrow$ for years

$\rightarrow$ this will result to Ignorance of resp center to $\uparrow \text{CO}_2$

"Down regulation" $\rightarrow$ No longer a stimulant for Resp center

$\rightarrow$ Shifting of Stimulator " $\downarrow \text{O}_2$ Hypoxia" = Hypoxic drive

So Pt with COPD C/I to give high concentrations of

O_2 b/c $\rightarrow$ inh resp center $\rightarrow$ Cyanosis & Death!

COPD $\rightarrow \text{O}_2$ concentration 24% - 28% "Low" by venturi-mask

$\rightarrow$ target is 90% O_2 Saturation" (hypoxia)

if 92% $\rightarrow$ resp depression

PaO_2

80 - 100 mmHg

Pressure measured

by ABG

O_2 Saturation

$> 92\%$

Percentage of Hb sat by O_2

measured by Pulse oximeter

Resp regulation

Chemical
↓

Central
"Brain stem"
($\uparrow \text{CO}_2$ - $\uparrow \text{H}^+$)



Pressure
↓

Peripheral
"Baro-receptors"
in Arch of aorta
& Carotid sinus
($\downarrow \text{PaO}_2 < 80 \text{ mmHg}$)



Alveoli



Patent

Collapse

- | | | |
|---|---|---------------------------|
| 1- Phospholipid "Surfactant" | → | 1- Amniotic fluid |
| 2- Elastic fiber destruction 'elastase' | → | 2- Elastic fibers |
| 3- -ve pressure of Pleura | → | 3- +ve pressure of Pleura |
| 4- Residual volume | → | |

1) Fluid & Phospholipid



Intra-uterine → amniotic fluid get in → Surface tension $\uparrow$ → help Collapse

at 32 wk gestational → Production of surfactant



" Phospholipid = head = hydrophilic
tail = hydrophobic

→ $\downarrow$ surface tension → Prevent Collapse

2) Elastic Fiber & Elastase

Normally there is balance b/w elastic production & Destruction

Pt with emphysema $\rightarrow$ break in elastic fiber $\rightarrow$ Permanent dilation "Hyper-inflated Chest"

3) Pleural Pressure

Intra Pleural pressure always -ve "-1 to -3"

During inspiration $\rightarrow$ Parietal & visceral become away from each other

$\uparrow$ negativity "-3"

b/c cage become bigger $\rightarrow$ Pull visceral

away from Parietal $\rightarrow$ -ve

"Help air get in"



During expiration $\rightarrow$ Cage get smaller

$\rightarrow$ Parietal & visceral become close together $\rightarrow \uparrow$ Pressure

$\rightarrow$ but still negative "-1" air get out

So -ve Pressure keeps alveoli patent

if Pleural Pressure +ve "tension pneumothoracic - Pleural effusion"

$\rightarrow$ Collapse

"All Pleural disease" $\rightarrow$ Collapse

Resp disease

Obstructive

airway disease

- 1 = Bronchial Asthma
- 2 = COPD
- 3 = Bronchiectasis
- 4 = Bronchiolitis Obliterans



(Difficult in expiration)

Restrictive

lung disease

- 1 = ILD
- 2 = Obesity
- 3 = ms weakness
↳ GBS, MG
- 4 = Skeletal Deformity



(Difficult in inspiration)

normal in expiration
upper airway → Dilation
lower airway → narrowing



Pt Problem in Lower airway

or OAD



at inspiration b/c wider air get in
but expiration → narrow by 2 factor
physiological & Pathological



wheeze more in
expiratory phase



normal in inspiration
upper air way → narrowing
lower air way → Dilation



Pt Problem in upper
airway



at inspiration b/c
narrow → + disease
narrowing more ex.
Foreign body



Stridor "inspiratory
saw"

Both Cause Cough & Dysnea

OAD $\Rightarrow$ Cough presenting symp

b/c cough receptor in mucos of airway

any irritation $\rightarrow$ stim Cough

Restrictive $\Rightarrow$ Dysnea presenting Symp

b/c problem in parnchyma "ex: Fibrosis"

$\rightarrow$ alveolar shrinkge $\rightarrow$ air can't get in

$\rightarrow$ Dysnea

"wheeze" only in OAD b/c narrowing of airway

but restrictive $\rightarrow$ airway are normal

X-ray

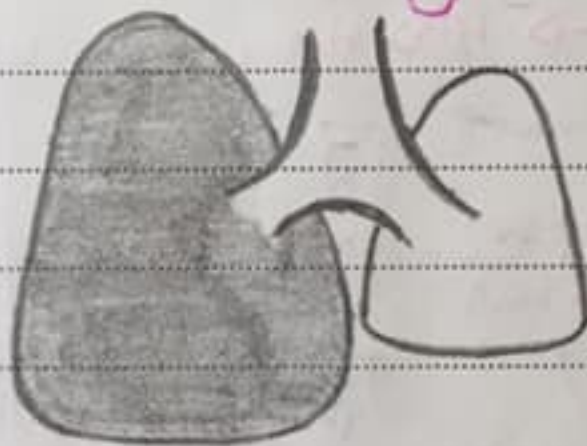
OAD

lung bigger

air in but

can't get out

$\rightarrow$ Black x-ray



restrictive

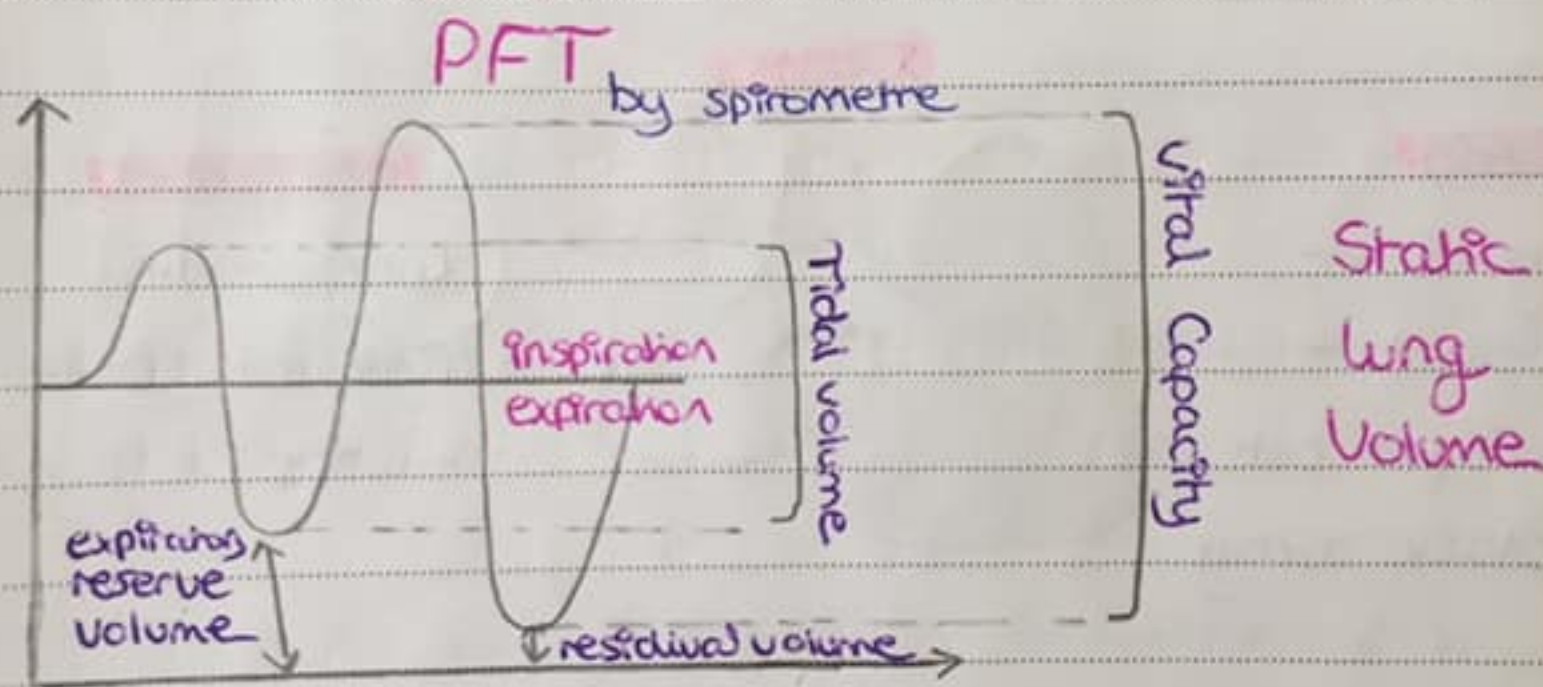
lung smaller

air can't get in

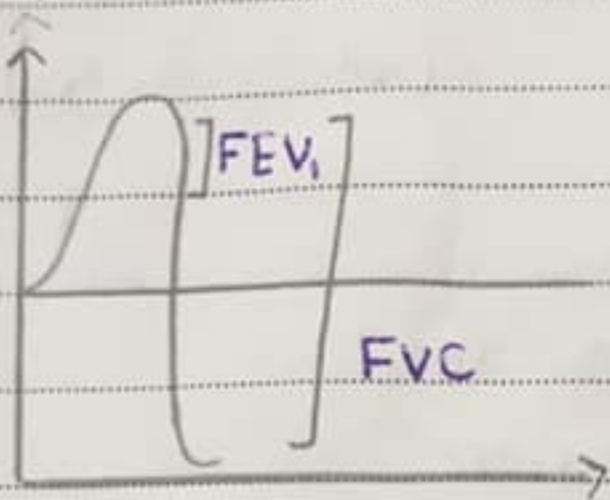
$\rightarrow$ white x-ray

* Respiratory inv *

- 1- CXR
- 2- ABG
- 3- PFT
- 4- CT Scan $\rightarrow$ Conventional - High resolution HRCT
- 5- U/S $\rightarrow$ most accurate in Pleural effusion
- 6- Blood Culture $\rightarrow$ infection
- 7- Throat Swab $\rightarrow$ URTI
- 8- Tuberculin test $\rightarrow$ screening for TB
- 9- Skin Prick test $\rightarrow$ for Allergy



- Tidal volume = amount of air expired after normal respiration
- Vital Capacity = amount of air expired after Deep Full respiration
- Residual volume = amount of air in lung after Full expiration
- expiratory reserve volume = amount of air in lung after normal expiration
- Total lung Capacity = V.C + RV



Dynamic

Lung

Volume "Time"

Forced Vital Capacity = amount of air expired forcibly & rapidly after full inspiration (FVC)

Forced expiratory volume at 1st second (FEV₁)

(FEV₁ ↓↓ significantly in OAD, slightly ↓ in restrictive)

b/c Obstructive main difficulty at beginning → Prolonged expiration

Ratio FEV₁ / FVC normally $\geq 80\%$

OAD $< 70\%$

- restrictive $> 70\%$ but $< 80\%$

FEV₁ = 80% of predictive

What is predictive?

According to 3 Parameters 1 = Age

2 = Sex

3 = height

ex: Mickleal 8 age 30yrs

(دراسة في UK)

♂

183 cm

$FEV_1 = 4L$

$FVC = 5L$

→ "Predictive"

Libyan Pt 8 age 30yr

♂

183 cm

} same Parameters

ex: $FEV_1 = 3L$

$FVC = 4L$

→ to be normal must be

80% of "mickleal" = Predictive

$\frac{3}{4} = 75\% \rightarrow$ less than 80% = Abnormal

So DIFF btw FEV_1 & ~~ratio~~ ratio btw FEV_1 / FVC

$FEV_1 = 70\%$ of Predictive "normal 80%."

"in comparison with a healthy

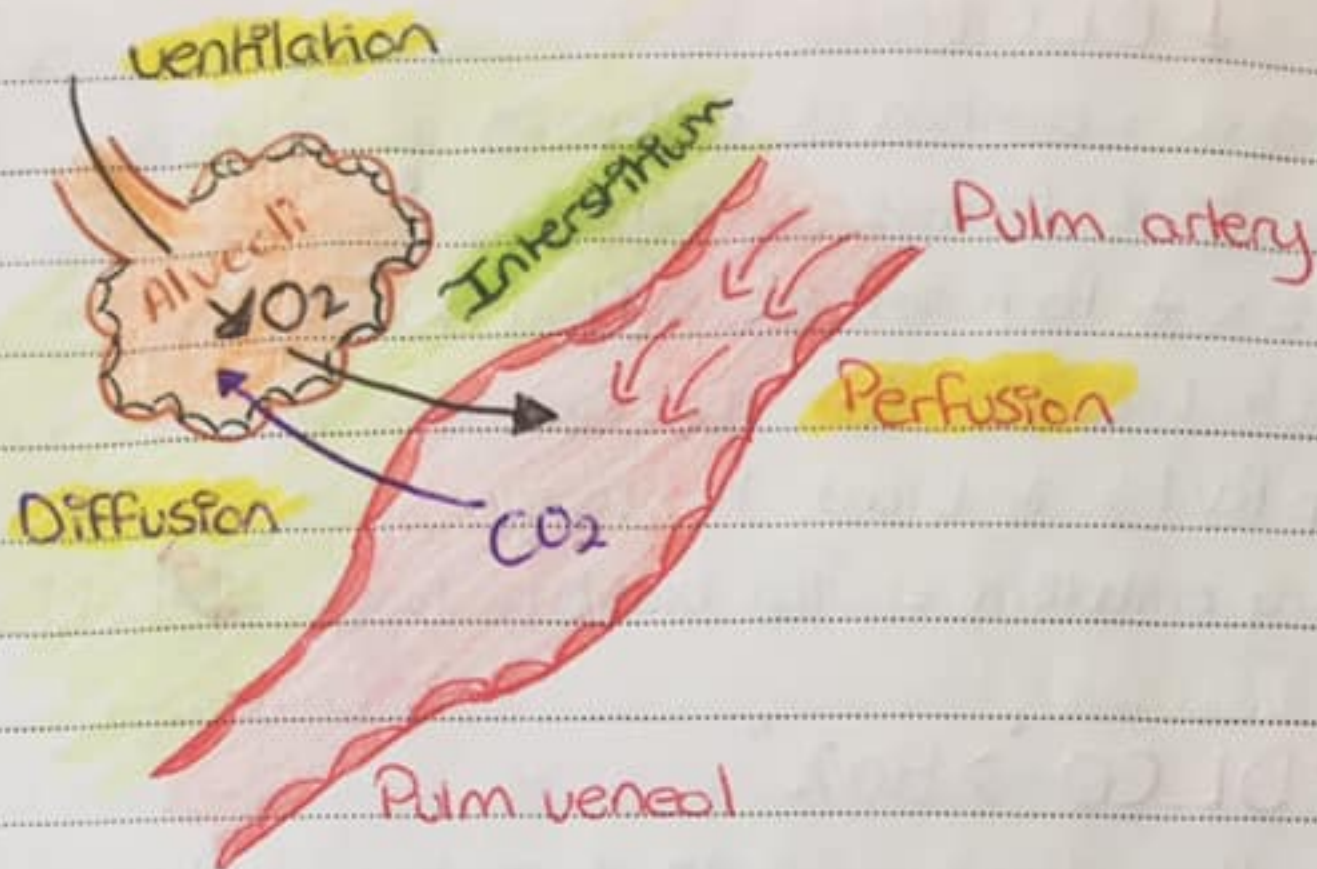
individual in U.K of same age / sex / height

$\frac{FEV_1}{FVC} = 75\%$ 'value of same Pt' 'normal 80%'

Note Obstructive & Restrictive $\Rightarrow FEV_1$ below 80%

but Ratio FEV_1 / FVC DIFF btw restrictive

& Obstructive



• For Normal Diffusion $\rightarrow$ Normal alveoli

Normal Interstitium

Normal Capillary bed

How to know that $\Rightarrow$ By DLCO

"Diffusion lung Capacity of Carbon monoxide"

Pt is given a known volume of CO "0.3" inhaled then exhaled

"Normally 80% is inhaled & 20% is exhaled"

So Normal (DLCO = 80%)

if Pt exhaled more than 20% $\rightarrow$ that means less than 80% are diffused $\Rightarrow$ DLCO $\downarrow$

DD of $\downarrow$ DLCO " $< 80\%$ "

- 1 = emphysema = dilation & destruction of Alveoli 
- 2 = ILD = thick fibrous interstitium 
- 3 = Vasculitis = $\downarrow$ Perfusion b/c inflam $\rightarrow$ narrowing
- 4 = Anemia = Low Hb for gas exchange
- 5 = HF "esp RVF" = $\downarrow$ Blood to lung
- 6 = Pulmonary embolism = Stop blood to lung 

DD of $\uparrow$ DLCO " $> 80\%$ "

- 1 = Alveolar Hge = ex: vasculitis & rupture of BoV
 $\rightarrow$ all blood in alveoli "No barrier" $\Rightarrow$ all CO + Hb
- 2 = Acute Attack of Bronchial Asthma
 $\rightarrow$ bronchospasm $\rightarrow$ air in but difficult to get out
 $\rightarrow$ time of diffusion $\uparrow\uparrow \rightarrow \uparrow$ DLCO
- 3 = Polycythemia = $\uparrow$ Hb $\rightarrow$ more Affinity
- 4 = Exercise

Note Chronic Bronchitis DLCO not effected

Case 35yr Libyan σ^7 presented with Chronic Dysnea & Cough

PFT shows &

$FEV_1 = 65\%$ of Predicted

$FEV_1 = 2L$

$FVC = 4L$

$DLCO = 70\%$

• $\frac{FEV_1}{FVC} = \frac{2}{4} = 50\% \Rightarrow$ Obstructive

• $DLCO \downarrow =$ emphysema

" $DLCO$ normal in Bronchial Asthma + Ch-bronchitis"

Case Pt Presented with Chronic Dysnea & Cough

PFT shows

$FEV_1 = 3L$

$FVC = 4L$

$DLCO = 80\%$

$\left. \begin{array}{l} FEV_1 = 3L \\ FVC = 4L \end{array} \right\} \rightarrow \frac{3}{4} = 75\%$ so Restrictive

$\rightarrow$ normal

Which of following could show this data?

- (A) B. Asthma (x) b/c Obstructive
- (B) IUD (x) b/c $DLCO \downarrow$
- (C) Obesity (x) Doesn't present with Cough "Only Dysnea"
- (D) M.G (✓) b/c restrictive + $DLCO$ normal

Dysnea + Cough b/c recurrent Chest infection

ABG

↳ For any emergency - severe Dysnea
taken by heparinized needle from any artery 'ex: radial'

Normal

$$PH = 7.35 - 7.45$$

$$PaO_2 = 80 - 100 \text{ mmHg}$$

$$PaCO_2 = 35 - 45 \text{ mmHg}$$

$$HCO_3 = 18 - 22$$

Resp Failure

$$= PaO_2 < 60 \text{ mmHg}$$

" $< 80 = \text{Hypoxia}$ "

Type 1

$$PaCO_2 < 50 \text{ mmHg}$$

Type 2

$$PaCO_2 > 50 \text{ mmHg}$$

"Hypercapnia"

b/c $O_2 \downarrow$

(Tx $O_2 \uparrow$)

High

Concentration

Up to 60%

by Polymask

Low

Concentration

18% - 24%

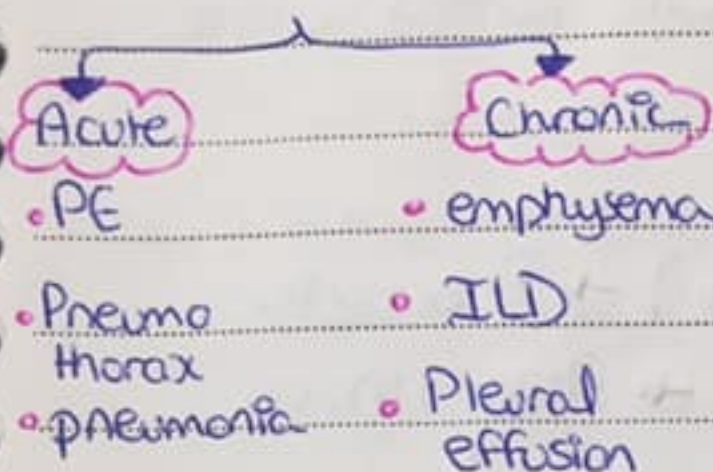
by venturi mask

b/c Hypoxic drive

Causes

Type I

any disease in resp
rather than II



Type II

- 1- Resp Center Depression "Pons/medulla"
↳ CVA - narcotics
- 2- ms Problem
↳ GBS - MG - muscular dystrophy
- 3- Skeletal Deformity
Kyphosis - Scoliosis
- 4- Chronic Bronchitis
- 5- Asthma "Near to fatal"

Note

- Any case emergency $\rightarrow$ $\text{CO}_2 \uparrow$ or $\text{pH} \downarrow \rightarrow$ Immediately next step = Intubation & M.V.
- $\uparrow \text{CO}_2 \rightarrow \text{VOD}$



$\uparrow \text{ICP} \rightarrow$ headache + papillP-edema
↳ Flapping tremor



$\rightarrow$ warm hand

large volume pulse "Bounding pulse"

Palmar erythema

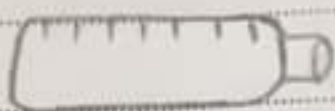
$\rightarrow \uparrow$ Permeability of BoV $\rightarrow$ edema

Note

High Concentration of $O_2 > 60\%$ more than 3 days "72 hr" is toxic to cilia $\rightarrow$ destruction
So Never give more than 3 Days
& when given $\Rightarrow$ humidified with normal Saline

Note (Key words)

- RF type I + (Fever - Cough - Chest pain) $\rightarrow$ Pneumonia
- Chest pain peripheral + $\uparrow$ with resp $\rightarrow$ Pneumothorax
- Uni L.L edema + (Cough - Dysnea - hemoptysis)
+ RF type I $\rightarrow$ Pulmonary embolism



Peak Flow meter

normally Peaked expiratory flow rate PEFR = 80% of Predictive value according to 1 = Age

2 = Sex

3 = height

Adv $\Rightarrow$ Portable - Cheap - given at home

used to monitor diseases ex: Asthma

Airway normally $\rightarrow$ Day Dilated "b/c Cortison $\uparrow$ / symp"
 $\rightarrow$ night Constricted "Cortison $\downarrow$ / parasymp"

b/c Bronchial Asthma is ① reversible ② Day & night variability $\rightarrow$ PEFR used in monitoring the disease

but in COPD airway are always narrow & No Day and night variability $\rightarrow$ PEFR can't be used for monitoring.

Def of Asthma :

Chronic inflammatory airway disease Characterized by hyper responsiveness to variety of external stimuli that lead to reversible narrowing in Lower airway

Cough
Dysnea wheeze → early in expiratory (big narrowing of lower airway)
→ late even expiratory

Trigger Factor "if Aevent => No need for Tx"

1 = Allergen = Ag Stim hypersensitivity type 1

→ ex: Dust mite "Dermatophagoid"

→ on Furniture lives on epiderm that falls from our body daily

"Attack ↑ night"

- Pollens "at spring"

- Pets "cats - rabbits - birds"

- Perfumes - Aerosoles

- Cockroach ==

2 = Occupation = ex: Farmer

↑ work days - ↓ holidays

Tx Change occupation

3 = Infection = viral → Children "RSV"

→ Adult "influenza - Parainfluenza"

"Not Bacterial"

4 = Drug induced = B-Blocker "Bronchospasm"

Aspirin or NSAIDs

5 = Exercise → Due to inhaled Dry air
 ↳ except 'swimming'

6 = Exposure to Cold → winter - Cold Drink

7 = Emotional Stress

Epidemiology → Children (70%) ♂ > ♀
 ↳ Adult (30%) ♀ > ♂

- Bimodal distribution

- more in Black

Note

• Bronchial Asthma ↑↑ Nowadays

• Peptic Ulcer ↓↓ b/c eradication therapy

Types of Asthma

1 = Extrinsic Asthma

♀ + ♂ → got married "consanguinity"

→ had a baby boy ♂ with mutation in Chromosome 11

that give (Atopy gene) = Family hx of Atopy

this gene is from his parents → so extrinsic!

- on his 1st exposure to trigger factor "dust" Ag

→ Pt had mild Cough + Dysnea + Production of high

titer of IgE "b/c genetically susceptible"

→ Abnormal activation of T-lymp → stim B-cell

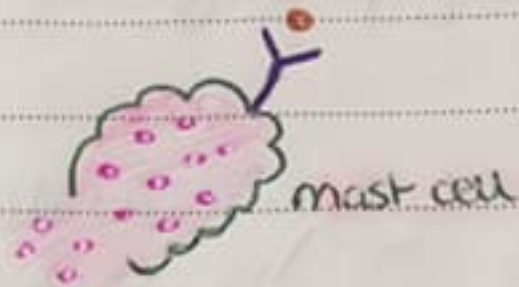
→ ↑↑ IgE on mast cell



On 2nd exposure "Ab ٥١" =
→ severe Attack

Ag - Ab → reaction on eosinophil & mast cell
"Dust + IgE"

↓
Degranulation



↓
release of all Chemical substance
"Histamin - serotonin - Bk"

↓
that Cause

↙
Bronchospasm
"Dysnea"

↘
↑ secretions
"Cough"

Note

Atopic "↑ IgE" could effect

- skin = Atopic dermatitis
- eye = Allergic Conjunctivitis
- nose = Allergic Rhinitis
- sinus = Allergic Sinusitis

Extrinsic Asthma more common in Childhood
good respond to Tx & Subsides by teenager group "most cases"

Risk Factor of Extrinsic "multi-factorial"

"Not all Pt that have Atopic gene will get Asthmatic Attack"

↑ Asthma

- Obese baby
- Supper hygiene theory
- ♀ Smoking during Preg or infancy
- Viral → RSV - Influenza - Parainfluenza

Protection

- Bact - Parasite
- Breast Feeding
- Anti-oxidants
"vit E - Selenium"

Supper-hygiene theory ↑ Allergy + Autoimmune
b/c immune system Not exposed to infection
→ hypersensitivity

So Bact + Parasite during Childhood Protect
from Asthma & hypersensitivity reaction

Note Smoking is a cause of Asthma & COPD

2 = Intrinsic Asthma

No family hx of Atopy

No gene mutation

IgE level normal "if elevated think of Parasitic infestation"

♀ Adult → infection by virus → after infection = hypersensitivity

Ag cause degranulation of mast cell & eosinophil

Directly without IgE stim

- No Atopy → Skin Prick test -ve
- Poor Prognosis - Not respond to Tx

Note Immunotherapy "monoclonal Ab" → effect IgE

Ab attach to IgE & prevent it's work

"So effective only in Extrinsic type"

Pathophysiology

→ (Child attack < 10yr old)

Dust get in → local reaction

→ IgE destruction cell wall of mast cell

Degranulation

Mast cell Granules

Contains

1 = Histamine

2 = 5HT serotonin

3 = Trypsin



Phosphorylation

lead to degranulation

Na⁺ Cromoglycate

"mast cell stabilizer"

inh degranulation

↓ release of substance

"So used as prophylaxis"

ex: before exercise

but NOT in acute

attack!

Histamine + Serotonin + Trypsin

stim. phospholipase enz

Phospholipid

Arachidonic acid

LOX

Leukotrienes

B, C, D, E

COX

PG

-ve

Aspirin

"Cystinyl leukotrienes"

sever Bronchospasm & ↑ secretion

(A) U.D of submucosal

B.V leading to swelling of mucosa

& Narrowing in diameter

(B) ↑ mucus secretion by goblet cell

(C) Smooth ms contraction & Spasm

narrowing

- Leukotrine antagonist $\rightarrow$ relief symp & Prevent attack
- Aspirin & All COX_1 inh are **C/I** in Asthma b/c
 $\uparrow$ leukotrienes "LOX Pathway" $\rightarrow$ sever bronchospasm
- Asthma is a reversible Condition, b/c all chemicals have a life time, when they are $\rightarrow$ Cleared from the body either spontaneously or by Tx

* **Clinical Presentation**

Cough - Dysnea - wheeze - Chest pain

"Not All 4 Symp should present"

$\rightarrow$ Some Pt present with Cough Only "Cough-variant" Asthma

Note

Persistent Cough after infection "without fever" $\Rightarrow$ think Asthma

"**Diurnal Variation**"

Symp in early stage "nocturnal attack" b/c

1 = $\downarrow$ Cortisol endogenous $\rightarrow$ Attack happen early morning

2 = Vagus over stim "Parasymp" $\rightarrow$ Bronchospasm

3 = Dermatophagoid "Dust mite"

Day free of symp only if exposed to trigger factor.

Seasonal Variation

$\uparrow$ in winter $\rightarrow$ Cold

$\uparrow$ in Spring $\rightarrow$ Pollens

Note

Asthma Completely reversible by bronchodilator in early Stage of disease

BUT in late stage smooth ms undergo remodelling & hypertrophy due to num of attacks $\rightarrow$ bronchospasm sever

"Dose that use to relief symp No more work $\rightarrow$ Need Larger dose "Partial reversible"

So Don't confuse btw COPD & Lat Stage Asthma

"By Hx ASK about symp before $\rightarrow$ if they where reversible or NOT!

★ Asthmatic Cough is Dry $\rightarrow$ if Productive 'Think infection'

Asthma

- Dry Cough
- Diurnal & seasonal variation
- reversible
- Hx of Atopy
- $\uparrow$ by trigger factor

COPD

- productive Cough
- No variation
- Fixed
- Hx of Chronic smoking

Sign

Inspection $\rightarrow$ $\uparrow$ RR, Pt distressed, recession

but usually Normal

Palpation $\rightarrow$ Chest expansion $\downarrow$ Bilat "During attack"

$\rightarrow$ TVF $\downarrow$ Bilat

$\rightarrow$ Trachea central

$\rightarrow$ Apex beat Not Palpable in sever attack

b/c lung Full of air

"TVF $\downarrow$ in all resp disease except Consolidation"

Percussion $\rightarrow$ Equal bilateral

Auscultation $\rightarrow$ air entry $\downarrow$ Bilat

$\rightarrow$ Type of Breathing

"vesicular breathing + prolonged expiration"

= 1st Change in Asthma

$\rightarrow$ Added Sound

- Rhonci all over chest "2nd Change"

- Crepitation if infection

Note

- Symp & Sign Normal "-ve btw attack"
- All finding are bilat + Scattered
- all OAD have vesicular breathing with prolonged expiration

- In Pediatrics Prolonged expiration is Normal b/c they have small amount of elastic fibers
So 1st Change is Rhonchi
- If No Rhonchi doesn't exclude Asthma in Adult b/c 2nd Change
- Inspection / Palpation / percussion Not imp in early stage b/c mainly normal → Auscultation is most imp

Pulsus Paradoxus

in acute attack → air trapped in → hyperinflated Chest
→ compression on heart lead to Pulsus paradoxus =

Drop of systolic BP more than 10mmHg during inspiration

DD



- Constrictive pericarditis
- Cardiac Tamponade



- acute severe Asthma
- Tension pneumothorax

Pulsus Paradoxus is a Poor Predictor sign for severity
→ many cases are severe & Don't have Pulsus paradoxus

Dx CXR Not used for Dx of Asthma

b/c usually normal btw attack and early stage of Disease

So -ve CXR doesn't exclude asthma

Inv

Exclude DD

Dx

PFT

PEFM

- CXR
- ABG $\rightarrow$ Resp failure 1+2
- CBC $\rightarrow$ Leukocytosis "WBC $\uparrow$ "
Diff leukocytic count = eosinophilia
- IgE kiter "Not specific"

PFT

during attack

$FEV_1 < 80\%$ of Predictive

FVC $\downarrow$

ratio $\frac{FEV_1}{FVC} = < 70\%$ "OAD"



(Reversibility test)

"Salbutamol 2 puffs"



measure after minutes



$FEV_1 \uparrow > 15\% = Dx$

Btw Attack

FEV_1

FVC

ratio

normal



(Provocation test)

exercise for 6 min or

Chemical by methacholine -

histamine Challenge test



$FEV_1 \downarrow$ worsening $> 20\%$
in chemical

& $\downarrow 15\%$ after exercise

Note

- If reversibility test Not improved $>15\%$ $\rightarrow$ other CAD
- If Pt tachycardia ($\uparrow$ HR) $\rightarrow$ can't give salbutamol b/c $\uparrow$ HR
 $\rightarrow$ give Prednisolone tab 30mg for 14 Days



if improved $>15\%$ = Dx

- Provocation test is Done with precaution b/c may go to sever attack of Asthma

- PD_{20} Provocation Dose 20 test
= Dose of Histamine that $\downarrow$ 20% about 16mg

PEFM if Pt normal PFT b/w attacks

$\hookrightarrow$ max velocity of air during expiration

Pt measure for 3 days in a wk for 2 weeks at morning & night

- take highest + lowest reading

Highest - lowest = Variability " $>20\%$ Dx Asthma"

• normal Pt = max 5%

• COPD = up to 10%
"NOT 20%"

- $PEFR = <80\%$ of Predictive

So PEFM used for Dx & monitor of Tx.

True or False

- Normal exam exclude Asthma (x)
- Normal Inv exclude Asthma (x)
- CXR useful for Dx (x)
- Pulsus Paradoxic good predictor for severity (x)
- Pt with intrinsic Asthma have $\uparrow$ IgE (x)
- Pt with Extrinsic Asthma have $\uparrow$ IgE (✓)

Note • IgE doesn't diff btw Extrinsic & Intrinsic Asthma b/c could be $\uparrow$ in Intrinsic due to Parasitic infestation.

- eosinophilia $\uparrow$ in both
- **So diff btw them by :**
 - * Skin Prick test " +ve = wheele reaction" in Extrinsic "Atopic"
 - * RAST "radio allergic Sorbent assay test" "Blood test to know allergy to which Ag"

Note Asthmatic Pt Not respond to Tx $\Rightarrow$ Do CXR

- to see if wrong Dx
- infection "TB - Fungal - pneumonia"
- Complicated to pneumothorax

Sameter's Triad = Widal Syndrome

1 = Aspirin induced Asthma "inh COX₁"

2 = recurrent nasal polyp

3 = recurrent Rhin sinusitis

Churg Strauss Syndrome

Autoimmune vasculitis + Bronchial Asthma
"Aseptic"

Any vasculitis $\begin{cases} \rightarrow \text{rupture} = \text{Palpable purpura} \\ \rightarrow \text{Thrombosis} = \text{ischemia} \end{cases}$

ass with sadal nose + sinusitis

ANCA +ve

Dx Clinically

Tx = Steroid + Cyclophosphamide

Note Bleeding subcut due to Plt dysfunction $\Rightarrow$ Flat purpura
but bleeding due to vasculitis $\Rightarrow$ raised purpura "Palpable"

Bronchial Asthma + Hemoptysis =

1 = Vasculitis "Churg Strauss"

2 = Infection "sever Cough"

"Bronchial Asthma NEVER present with hemoptysis"

Tx Asthma is treatable Not Curable

Target = 1. ↓ Symp

2. improve life style

3. minimal S/E of long life used Drugs

4. ↓ freq of Attack

Non Pharmacological

- Educate Pt about nature of disease
- How to use drugs - S/E - when response to Tx
- How to use PEFM "Full inspiration → then forced expiration"
- Avoid trigger factors
- "Salbutamol → must shake before use - Closed nose
Puff in half of inspiration.
- "Steroid Inhaler → Hoarseness of voice due to thickening of vocal
Cord
→ Cause Candidal infection
"Oro-pharyngeal + Oesophageal
So mouth wash after use

Pharmacological

Relievers

"Bronchodilator"

Preventors

Prevent attack

& if happen ↓ duration
of Attack

(A) Relievers

- **B₂ agonist** "1st line"
Inhaler / tab / I.V

Short acting
SABA

Salbutamol
Terbutaline

work 4-6hr

onset of work

min → max 15min

"used in emergency"

Long Acting
LABA

Salmeterol
Formoterol

work 12hr

onset of

work 15min

"Not used in
emergency"

mainly for
nocturnal symp

S/E Ⓣ ↓ K⁺

Ⓣ K⁺

b/c ↑ intracellular K

"Palpitation & Arrhythmia"

So in emergency when

given high dose of

Salbutamol Put Pt on

ECG monitor

& if severe hypokalemia

give I/v fluid + KCL

(when < 3.5)

② Fine tremor

"b/c sympathomimetic"

Theophylline

"tab + I.V" No inhaler

↳ Smooth ms relaxant of bronchi

by Phosphodiesterase inh

(CAMP)

(AMP)

When $\uparrow$ CAMP in cell

- Cardiac ms $\uparrow$ Contraction $\rightarrow \uparrow$ HR $\rightarrow$ S/E Arrhythmia
- Smooth ms relaxation

↓
So monitor by
ECG

"Fatal b/c narrow therapeutic index $\rightarrow$ toxicity
& has Drug-Drug interaction"
effected by enz inducers"

So NOT used for long term - mainly emergency

Anti muscarinic = Anti Cholinergic

- Ipratropium Bromide
 - Tiotropium Bromide
 - Oxitropium Bromide
- $\downarrow$ secretion mainly
• Broncho dilators
"Prevent Vagus Stim"

• Less effective b/c mainly $\downarrow$ secretion & Asthmatic
symp are mainly from sm.ms contraction

• S/E $\Rightarrow$ [even if given locally] $\rightarrow \uparrow$ IOP $\rightarrow$ Glaucoma
[buff can go to eye]

Note

B-Blocker C/I in Asthma by all routes
even eye Drop =

⑧ Preventers

Steroid "Local - Systemic"

- "Inhaler corticosteroid ICS" = Local

1 = Beclomethasone "Best"

2 = Budesonide → less systemic effect

3 = Fluticasone b/c not well absorbed

S/E → Hoarseness of voice - oropharyngeal candidiasis

Inhaler "local" Can cause Cushing Syndrome

& if Stopped suddenly cause Adrenal insufficiency

"↓ BP - epigastric pain - vomiting - etc"

- Systemic → Tab - I.V

- if given > 14 Day should ↓ gradually "Slow withdrawal"

- To stay in safe side use 7 days steroid "bang daily"

Can Stop suddenly → After risk for Adrenal Crisis

Leukotriene Antagonist

montelukast - Zafirlukast

Mast Cell Stabilizer

Na Cromoglycate "used before attack"

Omalizumab "Immunotherapy" "for Atopic Asthma"

Ab against IgE → ↓ Allergic reaction

Tx of Chronic Asthma

"Stepwise"

Step 1 (SABA)

S.O.S "as needed during day"

2 PUFF's at night



IF Not improved DON'T move to 2nd Step until U see if Pt is using Tx correctly, or Still exposed to trigger factor, wrong Dx or small dose!

Step 2 SABA + Inhaled Corticosteroid "2 PUFF's"

200

400

800

low dose

medium

"mostly start with" 400

Step 3 SABA + LABA + ICS

as needed

at night

2 PUFF's

Same dose Step 2

result

Good Controlled

Partial Controlled

Uncontrolled

Follow up

↑ dose of ICS
to 800

"Not respond
to LABA → remove"

SABA + ICS

+ Leukotrien Antagonist

or Theophylline

Step 4 "Trial Step"

(SABA + ICS "2000 = High dose"
+ leukotriene antagonist + theophylline)



Follow up for 1 month

if Not improved



+ Systemic B₂ against

"remove leuko or theophylline"

* LABA could be used in Step 4

* No role for Systemic steroid in Step 4

* IF Not improved After Step 4 think ->

Fungal infection "Allergic Bronchopulmonary Aspergillosis"
so must exclude

Step 5 Drugs Step 4 + Systemic steroid

"tab prednisolone"

must exclude Fungal before using steroid

if Can't exclude give course trial Tx "Itraconazole"

Antifungal before -> if improved = Proves Dx

Stay at Step 4

Note Systemic steroid best given at day time
b/c "Normally Cortisol low at night time"
if given at night $\rightarrow$ disturbance of Hypothalamic
Pituitary axis

S/E of Steroid $\rightarrow$ Osteoporosis

So give bisphosphonate as a prophylaxis
 $\Rightarrow$ inh osteoclast + $\uparrow$ Ca^{2+} deposition on bone

Note Down regulation "Stepwise reduction" if Pt stable
for 3-6 m & good controlled!

• Before exercise

Salbutamol

mast cell stabilizer

Leukotriene antagonist

} as prophylaxis

« Acute Asthma » emergency

"Rapid onset Dysnea - can't complete sentence"

Acute Severe Asthma	Life threatening Asthma	Near Fatal Asthma
PEFR < 55%	PEFR < 33	
HR > 110	b/c ms exhausted	RF type 2
RR > 25	& spasm to complete Obstruction	Hypoxia
Pt Can't complete one sentence in one breath		+
Pulsus Paradoxes	HR / RR / BP O ₂ < 60 mmHg CO ₂ < 50 mmHg 3C → cyanosis confusion, coma	CO ₂ > 50 mmHg
Rhonchi ↓ insp & exp	Silent ↓ Chest	

main Tx = M.V

(So presentation of Rhonchi doesn't indicate severity)

Monitor of Acute Asthma

- 1 = Vital sign "every 15 min"
- 2 = PEFR "to know type"
- 3 = Pulse oxymetry → to see O₂ Sat
- 4 = ABG → every 0.5 - 1 hr
- 5 = ECG → S/E of medication

Management of Acute severe Asthma

together given

(O₂ + SABA) "by Nebulizer"
High concentration
60% polymask
→ Salbutamol ⇒ 5mg / every 15 min
→ terbutaline ⇒ 10mg

⊕
I.V hydrocortisone 100mg - 200mg
or
Tab Prednisolone 40 - 50mg

Note if Pt in upper border of Acute severe → could go to life threatening (give I.V hydrocortisone)
(HR 130 / RR 35 / PEF 40%) (Tab Prednisolone)

⇓
wait 15 min

• Improved ✓

↳ 1st improved O₂ Sat

• Not improved

↳ O₂ < 92%

⇓

(Ipratrobium 0.5mg + SABA continuous)
SAMA "5mg / every 15min
or 10mg / every 1hr"

↓
IF Failed

I.V MgSO₄ "1.2-2gm" over 20 min
"Sm.ms relaxant"

↓

IF Not improved

"I.v B₂ Against or theophylline"

↓

if Not improved

M.V + Call senior

Poor prognosis

• IF Not improved ⇒ CXR

to exclude other disease "pneumothorax - Infection"

• Don't Delay Pt from M.V for x-ray! (Need Ab AntiFungal)

• once CO₂ ↑ → near to fatal → M.V

Chronic Asthma

Sign

	FEV ₁	PERF → variability
I	80%	< 20% "normal"
II	80%	20-30%
III	60-80%	] > 30%
IV	< 60%	
V	< 60%	

"Bw day & night"

Symp	D	N
I Intermittent	< 2/wk	1/m
II Chronic mild	≥ 2/wk	≥ 2/m
III Chronic moderate	Daily	> 1/wk
IV } Chronic Severe	Daily	freq ↑↑
V }		

"Inv more accurate than Symp"

Case

PT Step 1 for 10yrs + controlled

→ one day go to severe attack → ICU

took Tx + improved

when Home → Never back to Step 1

→ Start Step 2 or more

"So Any PT had acute severe attack → never back to Step 1"

At emergency → at any step improved

⇓

under monitor for 24hr

+ SABAs every 4-6 hr

⇓

at home Systemic Steroid 50mg / 7days

⇓

Follow up & Put in Step

COPD

MCC is smoking, so ↑ world wide

4th Cause of Death - Progressive disease Not Cured

* Causes of Death

1st IHD

2nd Lung Cancer → caused by Smoking :-

3rd CVA

4th COPD → caused by smoking :-

* Def

Fixed airway Obstruction

Irreversible - not improved by bronchodilator

Chronic Bronchitis

Productive cough for

3 successive months

in 2 successive years

ex: 5-6-7 = 2017

1-2-3 = 2018

↓
productive cough
daily

Emphysema

Permanent enlargement

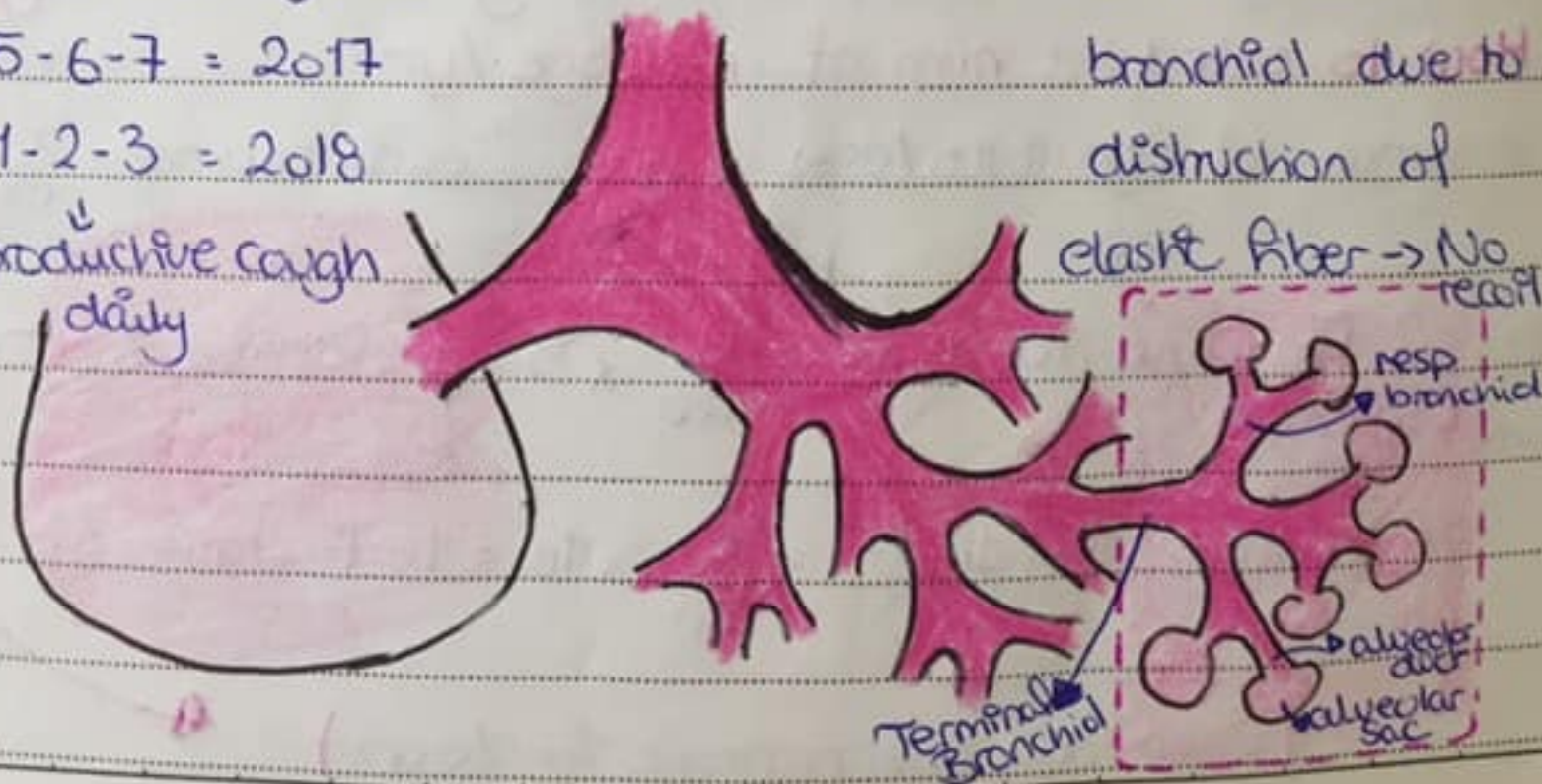
airway distal

to terminal

bronchiole due to

destruction of

elastic fiber → No recoil



Chronic bronchitis $\rightarrow$ pathology in main bronchus

So effect all lung together even if already functional normally air can't get out "more severe"

But emphysema effect part of lung & other parts normal "compensate" $\rightarrow$ then slowly progressive "less severe"

Note most cases are overlapped "mixed"
(Ch. bronchitis + emphysema)

Epidemiology 80

$\text{♂} > \text{♀}$ Age > 40 yrs

$\uparrow$ incidence b/c $\uparrow$ smoking

• Type of smoking

- Active smoker = Pt who smokes =
 - Passive smoker = sitting with active smoker
 - ex-smoker = Pt stopped smoking $\approx$
- ALL have risk COPD & lung Ca

• How to calculate num of package / yr

$$= \frac{\text{num of cigarette / day}}{20} \times \text{yrs} \quad \text{"each package = 20 cigarette"}$$

$$\text{ex: } 2 \text{ Package for 5 yrs} = \frac{40}{20} \times 5 = 10 \text{ Package / yr}$$

same risk if

$$1 \text{ Package for 10 yrs} = \frac{20}{20} \times 10 = 10 \text{ Package / yr}$$

(each day one package for 10 yrs)

Causes

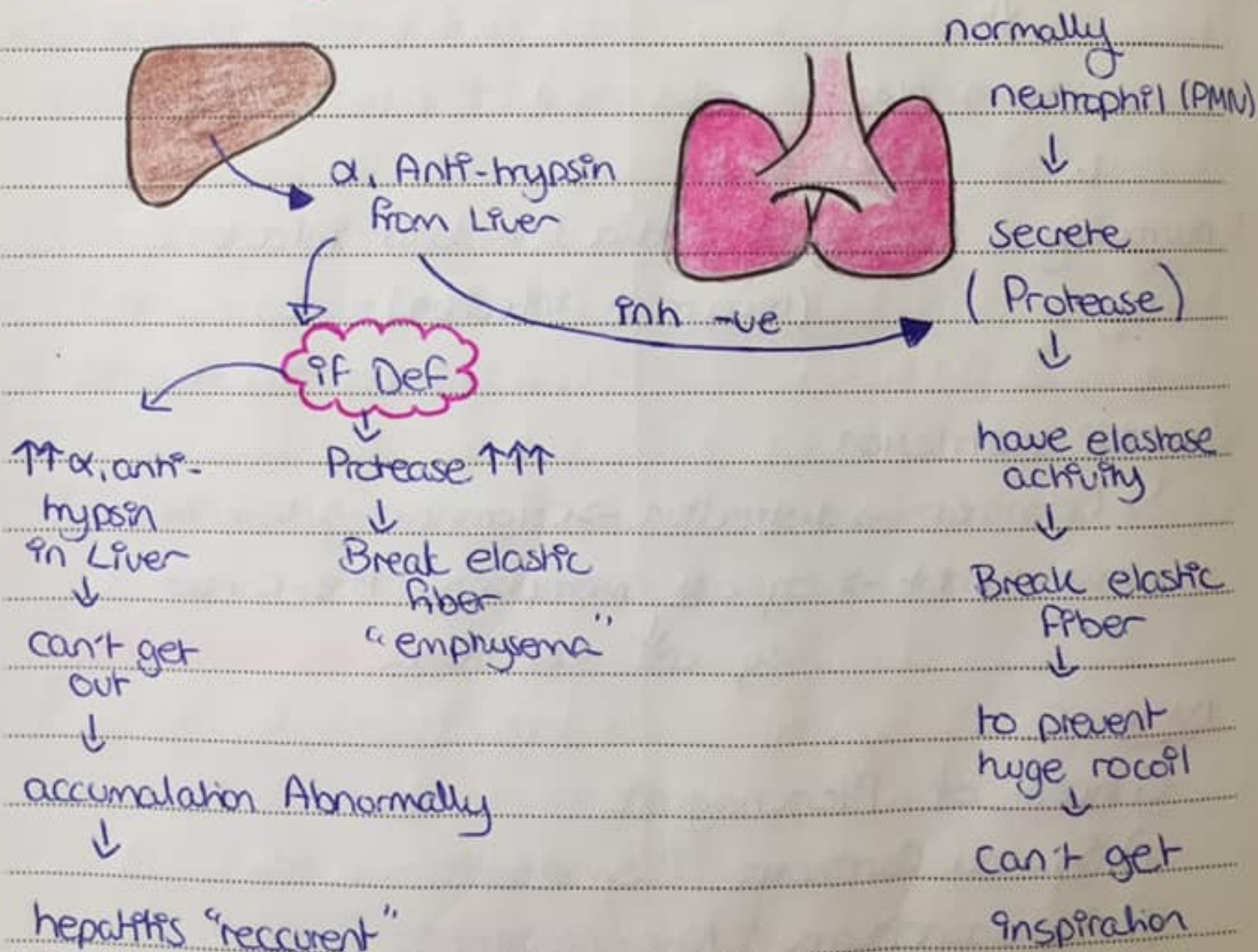
Smoking

- MCC "up to 99%"
- genetic risk
- 15% of smoker have COPD
- ↑ if 20 Package / yr (< 10 unusual)

α_1 Anti-trypsin Def
Less "1%"

present young < 40yr
AR "enz in
liver but can't
do exocytosis!"

(α_1 Anti-trypsin)



So think α_1 Anti-hypsin

- if COPD young < 40yr
- lung disease "emphysema" + Liver Stigmata "Jaundice"

Smoking $\rightarrow$ Ch. bronchitis

1 = Destruction of cilia

2 = Goblet cell hyperplasia + hypertrophy



$\uparrow\uparrow\uparrow$ mucus + No cilia move it



mucus plug



narrowing
air
way



good media for bact infection
(recurrent infection)

3 = epith^l destruction

$\hookrightarrow$ Columnar $\rightarrow$ destruction $\rightarrow$ Columnar $\rightarrow$ destruction

$\rightarrow$ at a point $\rightarrow$ Sq. cell metaplasia "Pre-Cancer"

Sq. cell carcinoma

Note

Cancer of Bronchogenic

1) Sq. cell Carcinoma "Smoker"

2) Adenocarcinoma "Non-Smoker"

Pt present with Cough from irritation of smoke

Dysnea b/c plug narrowing of airway

recurrent Chest infection

most
common
back
infection
in COPD

- 1 - H. Influenza "MCC" + exacerbation to acute
- 2 - S. pneumonia "MCC lead to pneumonia"
- 3 - Moroxella cattaral

• Rhonchi b/c mucus plug $\Rightarrow$ narrowing

$\hookrightarrow$ if Cough $\Rightarrow$ rhonchi $\downarrow$

• Creptation "inspiratory"

No Rhonchi or Creptation in

Ch. bronchitis

emphysema

Smoking $\rightarrow$ emphysema

• Stim elastase enz $\uparrow\uparrow$

• Inh α_1 anti trypsin $\downarrow\downarrow$

$\rightarrow$ emphysema

So Smoking mainly cause Ch. bronchitis

ass with emphysema

Risk Factor of COPD

1 = Low Birth wt

2 = maternal smoker

3 = air pollution "Cadmium"

4 = Child viral infection

MCQ

- All smokers will have COPD (x) 15% only
- COPD in smoker usually after 10yrs of smoking (x) > 20yr
- risk of lung Ca ↑ up to 70 After 40yrs smoking (✓)

Chronic Bronchitis



gas exchange
normally by
simple diffusion

at expiration → physiologically
narrowed airway + mucus plug
air-trapped in + $\text{CO}_2 \uparrow \uparrow$ in alveoli
with time CO_2 concentration ↑
intra-alveolar more than blood

↓
reverse diffusion

CO_2 from alveoli → Blood

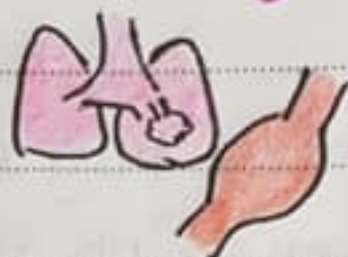
↑ CO_2 in blood → stim resp center

with Chronocphy → hypoxic drive

- $\text{O}_2 \downarrow$ b/c most of lung
effected ⇒ **RF Type II**

- Lung Collapse can happen
in sever cases of Complete
Obstruction

Emphysema



gas exchange
normally by
simple diffusion

b/c alveoli dilated & distructed

→ ↓ O_2 Diffusion

b/c CO_2 40 times more
permable than O_2 so

O_2 effected 1st, & CO_2

can get out b/c bronchi
patent "No hypercapnia"

hypoxia mild b/c not

all lung effected ⇒ Part
compensating

↓ O_2 , CO_2 normal = **RF Type I**

↓

Late Stage RF type II

So emphysema doesn't lead to Hypercapnia due to:

1 = CO_2 more permeable $\rightarrow$ diffused

2 = Patent airway "No Obstruction = No accumulation of CO_2 in airway!"

3 = Not all lung effected "Compensate"

Both have ventilation/perfusion mismatch

(V/Q mismatch)

• Chronic bronchitis $\rightarrow$ ventilation $\downarrow$
Perfusion normal

• emphysema $\rightarrow$ ventilation good
Perfusion $\downarrow$ b/c enlarged alveoli
 $\rightarrow$ Pressure on Capillary!

Note =

COPD are risk for bronchiectasis b/c
recurrent Chest infection

So

Chronic bronchitis

$\downarrow \downarrow \downarrow \text{O}_2 < 60 \text{ mmHg}$

$\uparrow \text{CO}_2 > 50 \text{ mmHg}$

RF Type II

emphysema

$\downarrow \text{O}_2$

No Hypercapnia

RF Type I

All Complications are more with Chronic bronchitis



→ V_oC → Pulm arterial → Pulm HTN
→ Rt vent hypertrophy → ♥ Failure
(Cor Pulmonale)

↓ O₂ → kidney → erythropoietin ↑↑ → ↑ RBC
"2ny Polycythemia"

→ ↑ viscosity → thrombos!

"So Anti-Coagulant have a role"

NOTE

- Polycythemia + erythropoietin normal ⇒ P_{hy}

- Viscosity ↑ by → ↑ cells or ↓ Fluid

So any pt with severe dehydration →

give Anti-Coagulant with I_v Fluid



→ V_oD systemic circulation



↑ ICP → headache "symp"

Papilli edema "sign"

Flapping tremor "Asterixis"

↳ DD RF type 2

Renal failure

Liver failure "Cirrhosis"

Phenytoin toxicity

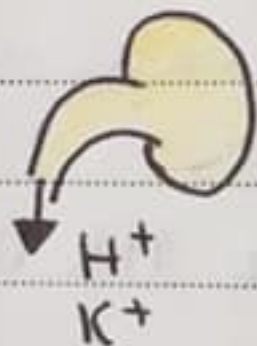
$\uparrow \text{CO}_2$ VoD of Peripheral

- large volume bounding pulse
- warm hand
- Palmar erythema

Note Clubbing Not Feature of COPD
if COPD Pt & had Clubbing indicate Cancer!

$\uparrow \text{CO}_2 \rightarrow$ acidosis " $\downarrow \text{PH}$ " = resp acidosis

Buffering System are "lung" + "kidney" to regulate PH
in resp failure type 2 $\rightarrow$ kidney compensate
"After Chronic period"



- $\uparrow \text{H}^+ + \text{K}^+$ in urine "acidic urine"
- Absorb bicarbonate HCO_3^- "Alkaline"
- + $\text{Na}^+ + \text{H}_2\text{O} \rightarrow \uparrow \text{volume}$
 $\rightarrow$ edema

So edema in COPD due to 2 Causes &

1 = Na^+ water retention "more"

2 = Rt $\heartsuit$ Failure

Note

• in ABG of RF type 2 $\rightarrow \text{HCO}_3^- \uparrow$ upto 40

indicate compensation of renal & Chronicity

• if HCO_3^- normal indicate acute or renal failure

$\geq$

Table to Diff btw Ch. bronchitis & emphysema

"Page 17"

Names

Emphysema = Pink Puffer

- Pink b/c polycythemia due to $\downarrow O_2$
- Puffer b/c part of lung normal $\rightarrow$ compensate & resp center Not effected.

Ch. bronchitis = Blue bloater

- Blue b/c cyanosis due to sever hypoxia
- Bloater b/c edema peripheral due to $\uparrow CO_2$

Built

emphysema thin

b/c compensation by normal part of lung $\rightarrow \uparrow RR$ "Dysnea"
 $\rightarrow \uparrow BMR \rightarrow \downarrow wt$

Ch. bronchitis = Obese

- due to edema
- No Compensation "tolerance to $\uparrow CO_2$ "

Cyanosis

emphysema less b/c compensation "Pink"

Ch. bronchitis more

Dysnea

emphysema $\uparrow\uparrow$ b/c Compensation

Ch. bronchitis less b/c Failure to compensate

ms weaker + tolerance + No normal Part
 $\downarrow$
due to airway obstruction

Hyperinflation

emphysema $\uparrow\uparrow$ b/c dilation of alveoli

Ch. bronchitis less

Cor Pulmonale

less with emphysema

more with Ch. bronchitis " $\uparrow$ JVP + lower limb edema

(also due to Na^+ H_2O retention)

RF

emphysema type 1 / Ch. bronchitis type 2

ABG

emphysema disturbance late when all lung effected

Ch. bronchitis early

Note

How to diff btw emphysema & Ch. bronchitis
by DLCO

↓ emphysema - Ch. bronchitis normal

NOT by ABG b/c RF type 2 can happen in
emphysema at late stage

C/P

- Cough → most common presenting symp
- Dysnea → by exercise tolerance test "in 6 min"
↓
How long can he go?
- Wheeze

Cough esp in Ch. bronchitis due to irritation of
Cough receptors in epithelium

Productive Large amount - Sticky sputum

White in Color "if yellow purulent = infection"

more in morning

Cough if persistent can lead to →

- Headache
- retinal detachment
- Hemoptysis due to injury of pharynx "streak of blood"
min in amount
- Chest Pain
- Abdominal hernia "reccurent"
- Varicocele of testis "varicose vein"

Note Any Cough lead to hemophysis "min amount"

Hemophysis NOT feature of COPD

but Lung Cancer "Pure Blood"

So Clubbing + hemophysis → think Lung Cancer

DD of Symp

B. Asthma + Bronchiectasis "OAD"

Heart Failure

COPD

Fixed Continuos

Productive Cough

more in morning "Day"

Hx of Smoking

B. Asthma

Intermeted symp

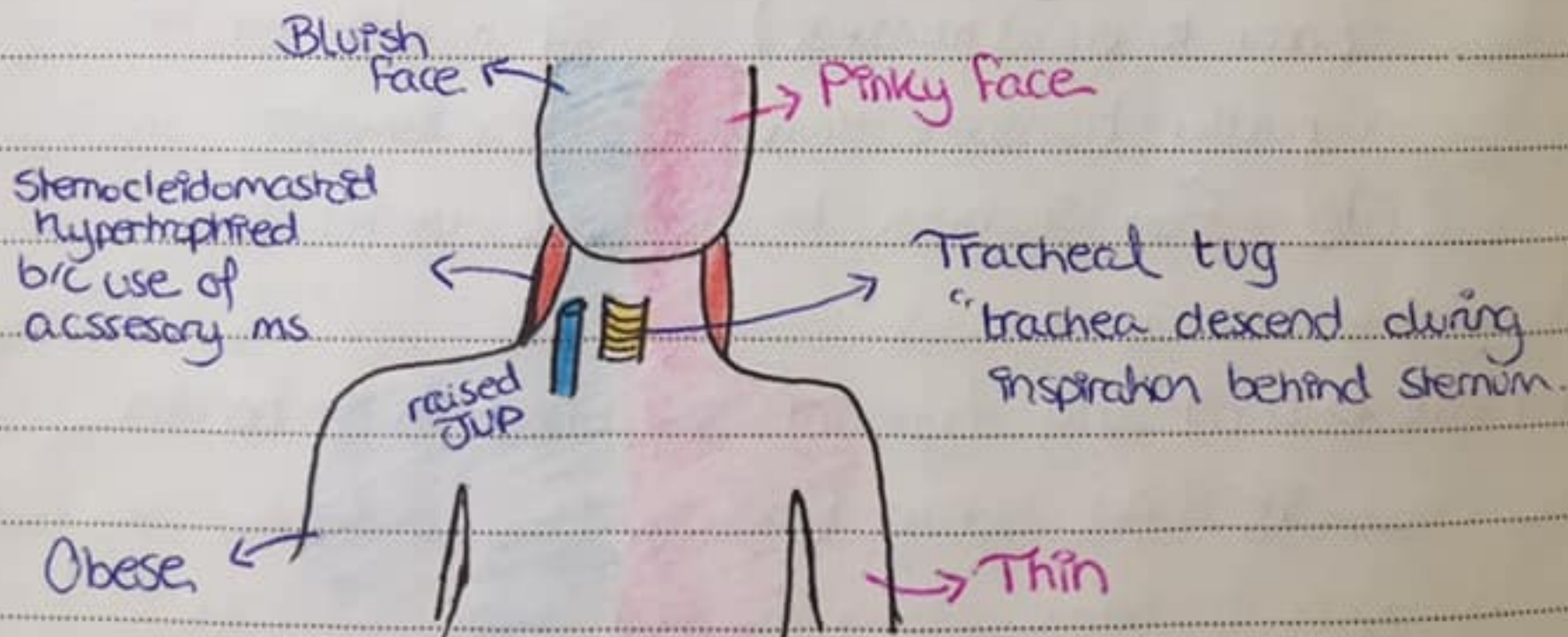
Day Cough

more at night

Hx of Atopy

Sign of COPD

"General"



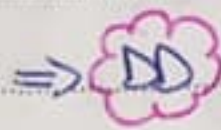


Palmar erythema

Asterixes

Bounding Pulse

Warm & Dry

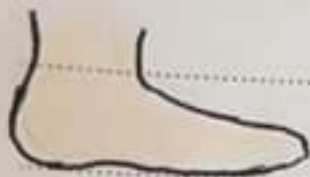


• warm + sweaty =

→ Thyrotoxicosis "↑BMR"

• Cold + Sweaty =

→ Symp over activity
"anxiety"



L.L edema

• Bilateral "♥ failure or Na⁺ retention"

↳ Diff by JVP → ↑ in HF

• Unilateral if DVT + PE

Local examination

Inspection

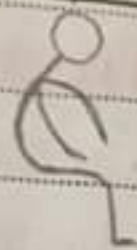
1) Barrel Chest → ↑ AP diameter "indicate obstruction of
(near to equal transverse) air + distressed"

"normally transverse diameter > AP diameter"

"AP → from Sternum to thoracic vertebra"

* NOTE ⇒ Don't say hyperinflated chest in inspection
'it's an x-ray finding'

2 = Sitting forward with pursed lips "fishy lip"
help air get out during expiration



3 = Distressed "RR ↑↑"

- using accessory ms → recessions → Suprasternal
(even sternomastoid) Intercostal
Subcostal

- acting ala nasi or breathing by mouth.

Palpation

1 = Chest expansion → ↓ Bilateral b/c ↓ airtentry
to lung "تضييق"

Note • Chest expansion is ↑ in Diameter from Full
expiration to full inspiration "normally > 3cm"

- all respiratory disease ↓ chest expansion

2 = TVF → ↓↓ "all resp disease ↓ except consolidation"

- normally voice moves in solid > fluid > air, why?

Solid → particles are near to each other, but air are far

- normal lung has "solid = parenchyma & air"

- any disease ↑ air "ex: CAD" → TVF ↓

but in pneumonia → ↓ air "dead space" + ↑ fibrosis + C.T

so TVF ↑↑, also lung Cancer or mass!

All Pleural disease ↓ TVF "تقييد" → multiple -ve sign

3. Trachea (Central)

Note • lesion bilateral $\rightarrow$ central trachea

- Unilateral $\rightarrow$ trachea deviated "Push or Pull"
"Fluid - air $\rightarrow$ Push" "Fibrosis $\rightarrow$ Pull"

except pneumonia if unilateral "Lobar" $\rightarrow$ central trachea

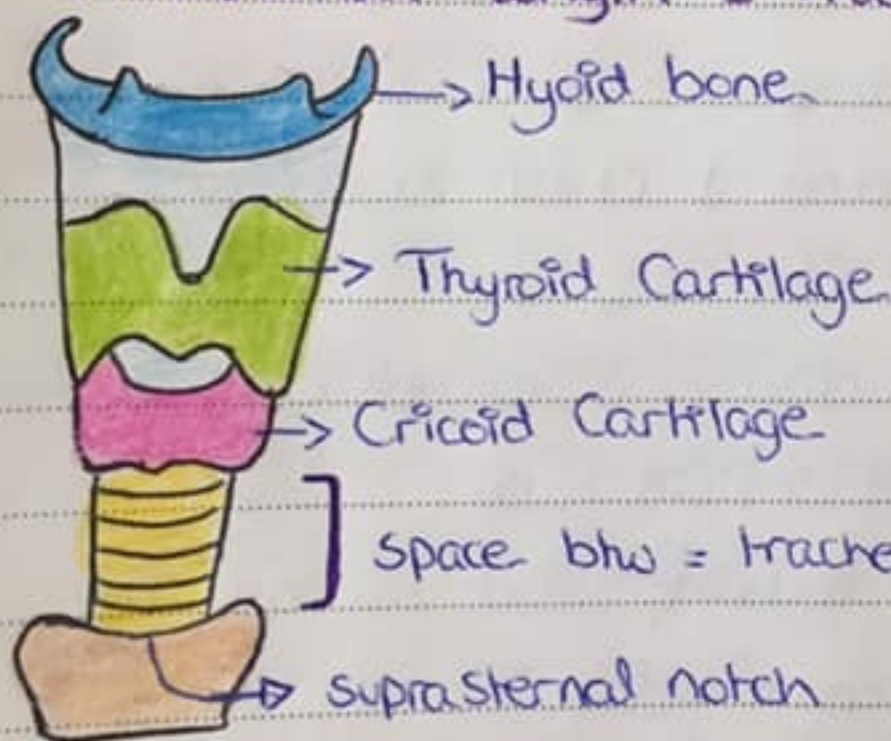
- Trachea = upper mediastinum

♡ = Lower mediastinum

if ♡ only deviated & trachea central $\Rightarrow$ CVS cause
but if both shifted $\rightarrow$ resp cause

- Tracheal tug $\rightarrow$ descend during inspiration

- Cricosternal length = Tracheal length



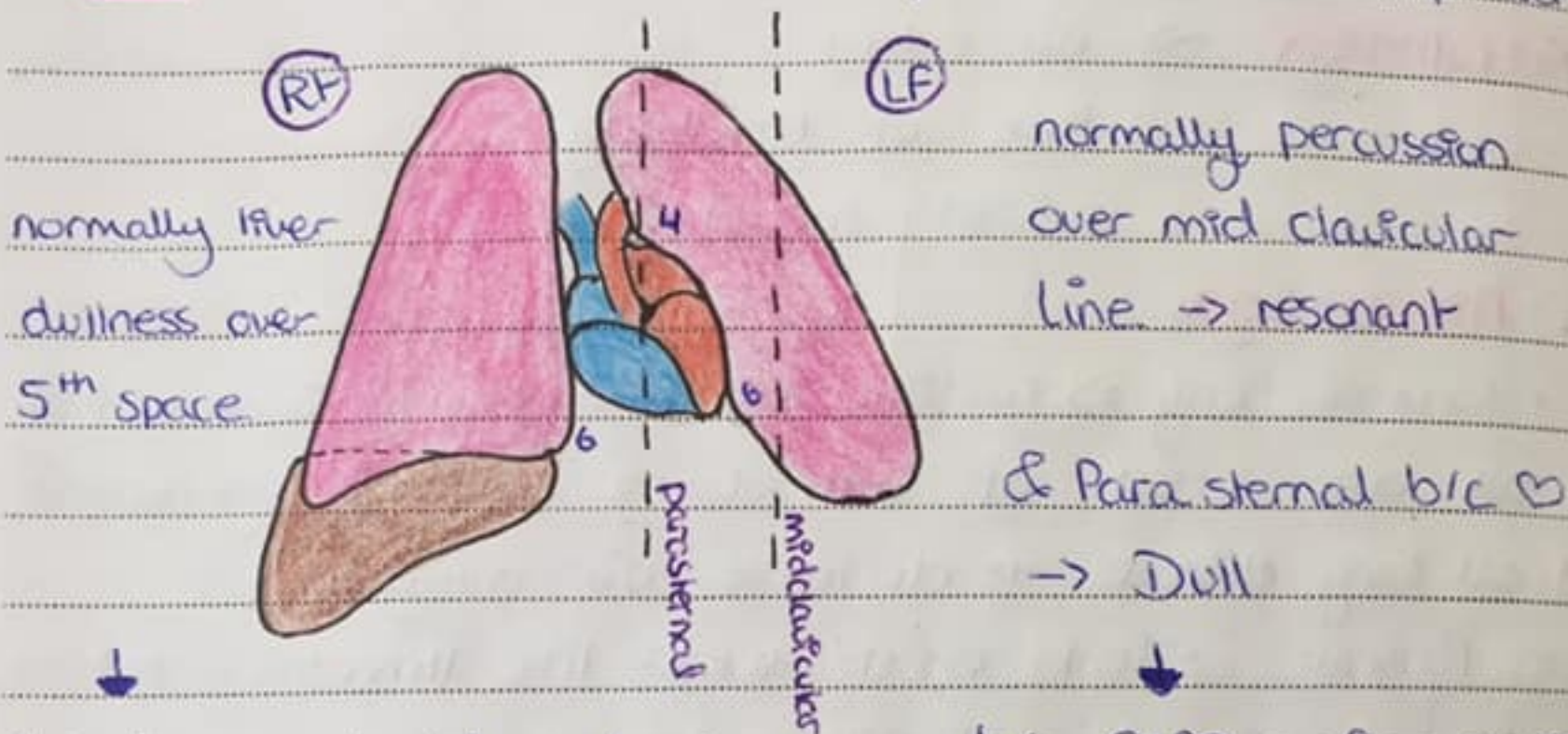
Shortening of palpable area of trachea

"normally 3 fingers"
3-4cm

b/c chest of COPD enlarged AP & vertical $\rightarrow$ suprasternal get up $\rightarrow$ cover space " $< 3\text{cm}$ " = Trachea Not well Palpable

Apex beat → Not palpable b/c air over ♥

Note In COPD ↓ dullness of ♥ + ↓ dullness of liver



normally liver
dullness over
5th space

normally percussion
over mid claviclar
line → resonant

& Para sternal b/c ♥
→ Dull

↓
b/c lung push Liver down
"Liver Ptosis = Pushed liver"

↓
b/c COPD → air over ♥
→ resonant

dullness over space 5 ↓

- Palpable liver with normal span

Key points for COPD

1 = Smoker

2 = Productive cough "white sputum"

3 = ↓ or Absent dullness of Liver & heart

4 = Apex not palpable

Subject: / /

Percussion → Hyper-resonant bilateral diffuse

↳ DD pneumothorax → mainly unilateral
Locally Apical

Auscultation → air entry
type of breathing
added sound

1) **Air entry**

- * Normal lung ⇒ audible bilateral equal symmetrical all over chest of both lungs "Don't say normal"
- + all lung disease → decrease air entry ↓
or Absent "silent chest" → ex: life threatening asthma
- Pleural disease "effusion" - Lobectomy
- No resp disease ↑ air entry

⇒ COPD → ↓ bi-lateral all over zones of both lungs

2) **Type of breathing**

vesicular breathing with prolonged expiration

3) **Added sound** → Ronchi = musical sound 2ry to narrowing of lower airway

"narrowing b/c secretion" Not bronchospasm

best heard during expiration

• Crackles → during inspiration & expiration "Biphasic"
↓ by cough "Coarse crackles"

↳ DD (Bronchiectasis)

* Added sound mainly in Chronic bronchitis NOT in emphysema.

NOTE

Pneumonia →

early Fine

Crackles

→ Fluid in

alveoli → at end of inspiration

Can't get out with Cough

"Persistent after Cough"



DD of Fine Crackles

- alveolar edema "congested lung" from LF ♡ Failure

- ILD b/c Fibrosis around alveoli → shrunken

when air get in → open alveoli → Crackles



Pneumonia → Late

with Tx & mucolytics

→ all secretion get out

"Coarse Crackles"

(resolution stage)

Inv for Complication

- CBC = Polycythemia
 - echo = Rt $\heartsuit$ Failure
 - RFT
 - LFT = α_1 anti-trypsin
 - Bronchoscopy & biopsy
 - CT - Scan
 - Percutaneous biopsy
- } inv for Cancer
if COPD + Clubbing + hemoptysis

Inv for Dx

CXR $\rightarrow$ early could be normal
 $\rightarrow$ late "Hyper-inflated chest"

1 = $\uparrow$ vertical diameter

2 = Ant ribs > 6

3 = Post ribs > 10

4 = tubular mediastinum

5 = Flat Diaphragm

6 = Black x-ray "b/c air $\uparrow$ " Bilateral
& $\downarrow$ lung marking of tissue

7 = $\uparrow$ intercostal space $> 1\text{cm}$ "normal 1cm "

ABG $\rightarrow$ RF type 1 = emphysema "early Not disturbed"
RF type 2 = Ch. bronchitis

Dx test PFT

$FEV_1 < 80\%$ of pre-dictive
 $FVC = \text{normal or slightly } \downarrow$
 $FEV_1 / FVC < 70\%$ } Like all OAD



give salbutamol

"No improvement $> 15\%$ " } Slight improvement
No reversibility } Fixed obstruction



DD COPD } Diff by imaging
bronchiectasis } (CT-scan)

Note Asthmatic bronchitis $\rightarrow$ case that has bronchial
Asthma + Ch. bronchitis "reversibility border line"

• How to Diff btw types of COPD? by DLCO
(if $\downarrow$ emphysema) - (normal = Ch. bronchitis)

Tx Not curable just treatable

lung transplant is only Tx (but Pt smoker "won't")
stop $\geq$

or α_1 anti-trypsin $\rightarrow$ new lung will effected by
deficiency!

Non pharmacological

- education about disease
 - harmful effect of smoking
 - How to use medication
-
- Tx improve prognosis "improve lung function"
 - 1 = STOP smoking
 - 2 = Long term O_2 therapy LTOT
 - 3 = lung volume reduction by surgery
-
- all these cause regression of progression of disease
 - Stopping smoking decrease decline of lung function & preserve lung function upto 80yrs
- After 80yrs Not much benefit of stop smoking

Medical Tx "Symptomatic" "improve life style"

Tx according to FEV_1

Best parameter of PFT to detect prognosis & Tx

FEV_1 80% or less = mild

50 - 80% = moderate

< 50% = severe

mild → SABA + SAMA "S.O.S"
moderate → LABA + LAMA
sever → LABA + ICS or Both
⑥ LAMA + ICS

Chronic
COPD

Tx NOT used in Chronic

- 1) Ab as prophylaxis b/c ↑ resistance of bacteria ↑
- 2) Systemic Steroid b/c S/E more than improvement
- 3) Aminophylline b/c toxicity with Long term.
- 4) Anti-tussive "Anti-Cough" b/c Cough protective mechanism "remove mucus & infection "clear airway"
- 5) mucolytic

Surgery "lung volume reduction surgery"

1 = Lobectomy → b/c Non functional

effect normal lung by Compression

Dilatation of alveoli may open in Pleura → recurrent pneumothorax "very common" - emergency need chest tube
So Lobectomy

Note

- pneumothorax more common in COPD than Asthma & bronchiectasis

• Thoracotomy scar thick emphysema "Lobectomy"

Long term O₂ therapy LTOT

15hr/day, Low concentration 24-28% b/c hypoxic drive
high pressure volume rate

by venturi mask or nasal cannula

LTOT ↓ Complication + ↑ prognosis "improvement"

Indication

PaO₂ < 55 mmHg

↓
(even 1 reading)

PaO₂ < 60 mmHg

⊕ one of following

1 = Polycythemia

2 = Cor Pulmonale

3 = Peripheral edema

4 = Pulm HTN

2 reading
3wk
apart

Note • mmHg = $\frac{\text{kPa}}{7.5}$ Kilopascal

$$\text{ex } 55 \text{ mmHg} = \frac{55}{7.5} = 7.33 \text{ kPa}$$

• kPa × 7.5 = mmHg

Target for LTOT

PaO₂ = up to 60 mmHg } subnormal b/c
& O₂ Sat = < 90% max } hypoxic drive

HIP vaccine

hemophilus influenza	} given in	1 = ♥ Failure
influenza virus anuvaly		2 = COPD
Pneumococcal vaccine		3 = SCA
		4 = Celiac
		5 = Splenectomy
		6 = DM

"all risk for encapsulated organisms"

Complication of COPD

- Pneumothorax
- recurrent pneumonia
- recurrent infection → Bronchiectasis
- Resp failure type 1 + 2
- Heart Failure "Cor Pulmonale" Rt side
- Polycythemia + thrombosis

Acute severe attack of COPD "emergency"

rapid onset dysnea	} relapse
Sever deterioration of Symp	
Decline of PFT	

main cause is infection "esp in winter" ex: H. influenza "acc"

Strept. pneumoniae - Moraxella catarrhalis

Other Causes → DVT-PE / Arrhythmia

Tx O_2 "24-28%" + Bronchodilators
"SABA + SAMA"

(monitor O_2 by Pulse oxymetre continuous
→ once it's 90% → Stop O_2 b/c if upto 92%
→ resp center depression)

+ Systemic Steroid → Hydrocortison I.V 100-200 mg
→ Prednisolone tabs 40-50 mg
or Both

+ Find underlying Cause → why Pt got acute attack

Ab given if - Fever > 38

- Purulent sputum "PUS"

- CRP ↑

- CXR Changes "Pneumonia"

give Amoxicillin

↓

if Pt can't "ex: sensitive to it"

↓

give macrolids "Clarithromycin - Azithromycin - erythromycin"

↓

if Pt can't "Pt ♥ Problem"

↓

give Doxycycline

if No improvement

give IV Aminophylline + mucolytic to release secretion

↳ Phosphodiesterase inh $\rightarrow$ $\uparrow$ CAMP $\rightarrow$ sm. ms relaxation

- Slight Stimulate respiratory center

• given bolus 'stat' in 20min Slow infusion 200-250mg then maintenance "microgram"

• b/c toxicity risk for Arrhythmia so Put on ECG monitor

if No improvement

• give NIPPV "non-invasive +ve pressure ventilation"

by tight mask

ex: CPAP "Continuous +ve airway pressure"

BPAP "Bilevel +ve airway pressure"

• + Doxopram $\rightarrow$ stim resp center

S/E $\rightarrow$ Convulsion

Arrhythmia

Agitation

(not used anymore)

(alternative if No MoV)

Note

O₂ Delivery

Non invasive

mask "CPAP"

Invasive

Tracheostomy

MoV

Summary of Tx

O₂ + SABA + SAM_A + Steroid systemic

↓ (No improvement)

Ab + Aminophylline I.V

↓ monitor (vital sign -
ECG - Pulse oximeter - ABG)

ABG

PH

< 7.35

but > 7.26

↓

NIPPV

"improve prognosis
& ↓ indication for
M_oV"

PH

< 7.26

↓

M_oV

S/E barotrauma

pneumothorax

infection

bleeding

Note

- Bicarbonate not used to Tx Acidosis
- Anti-Coagulant is used b/c Pt risk for thrombosis

Prognosis & mortality rate

By BODE index

B $\rightarrow$ BMI < 21 or > 21

O $\rightarrow$ Obstruction FEV_1

D $\rightarrow$ grade of Dysnea (1 $\rightarrow$ 4)

E $\rightarrow$ exercise tolerance "in 6min"

ex: Score 0-2 $\rightarrow$ 10% Die in 52m

7-9 $\rightarrow$ 80% Die in 52m

Hx of Dysnea 8

First ask about association to exclude systems

Time "onset - Duration - seasonal variation"

for all $\left\{ \begin{array}{l} \text{Localization} \rightarrow \text{case of pain} \end{array} \right.$

Quality

Severity

3A $\rightarrow$ Aggregation - Alleviation - Association

Dysnea
ass

+ cough
+ wheeze



DD resp (OAD) $\rightarrow$ symp together

$\heartsuit$ Failure $\rightarrow$ orthopnea / PND

GERD

Dizziness

Fatigue

Papillation

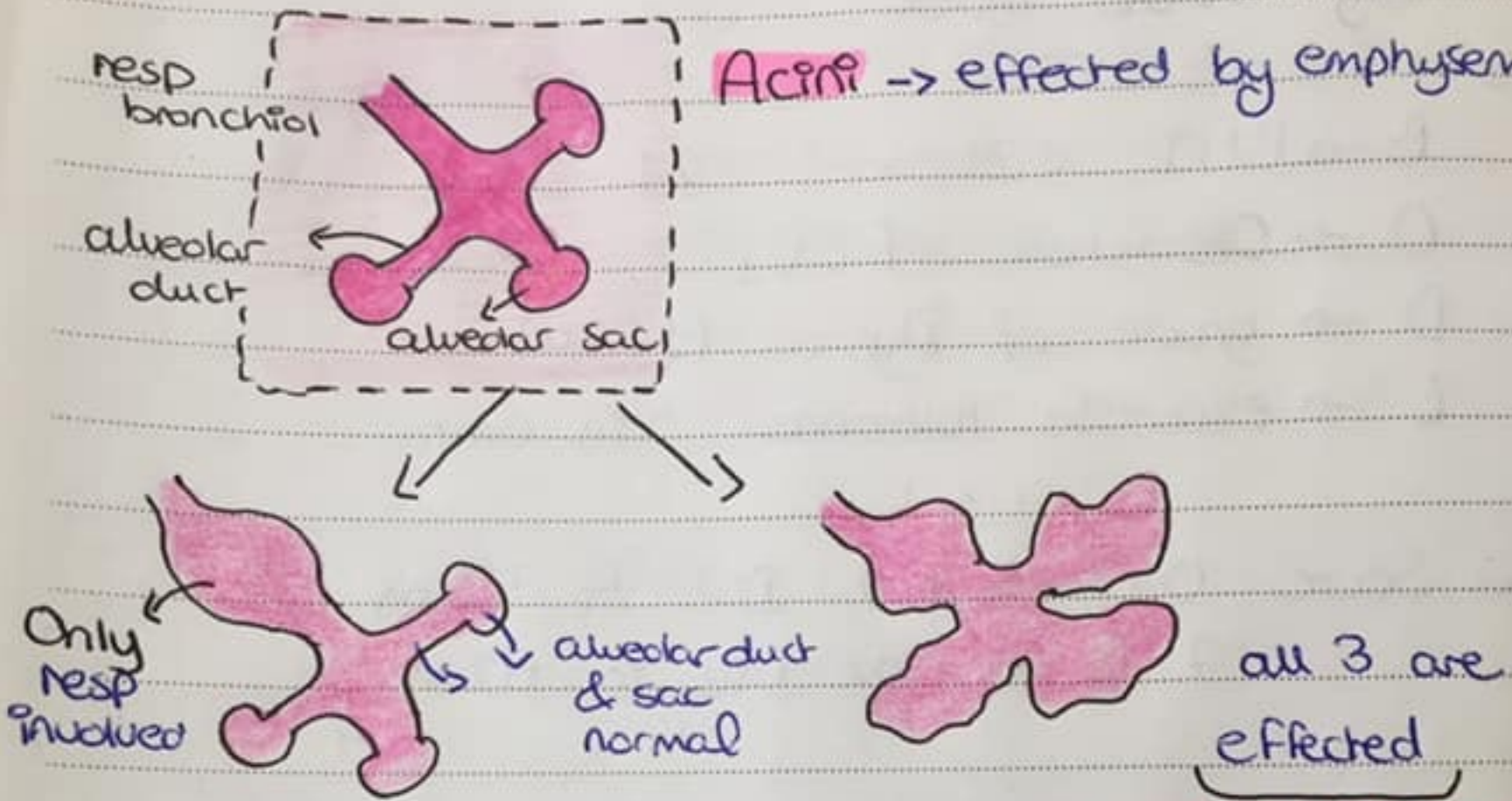
DD anemia

HF

Psychological

Note (Type of emphysema)

Acini $\rightarrow$ effected by emphysema



centro-acinar
emphysema

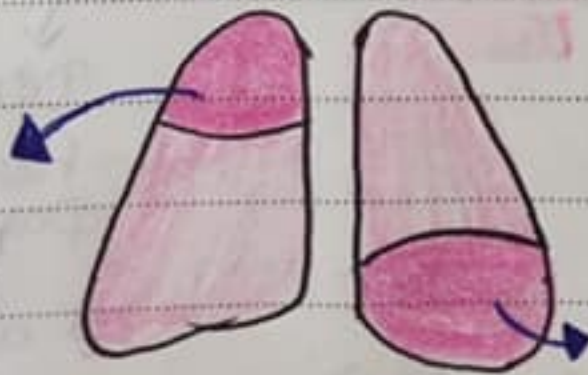
pan acinar
emphysema

mainly with smoking
b/c smoke reach resp
bronchiole 1st

with time progress to
pan acinar

mainly due to
 α_1 anti-trypsin Def
b/c disease systemic
effect all areas

Smoking mainly
effect upper zone
"Apical"
then progress

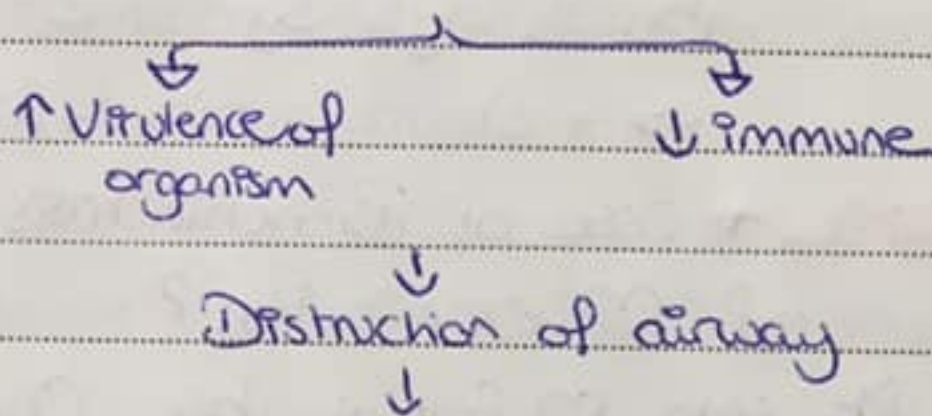


α_1 -Anti-trypsin
mainly effect lower
zone
then Progress

(Bronchiectasis)

Permanent dilatation of one or more bronchi due to Chronic infection that caused Chronic Inflammation & Destruction & Dilatation of airway

So most common cause = (Strong recurrent infection)



Irreversible dilation

Dilatation will effect normal alveoli & lung parenchyma & cause inflammation & consolidation to surrounding
→ Loss of normal lung Architecture

Causes → MCC = TB

- Idiopathic "40%" → if No cause found!
- Congenital

A) Cystic Fibrosis

AR - Deletion of Chromosome 7

cause thick secretion "mud like"

difficult to cough → mucus plug

B) Immotile cilia Syndrome

ex: Kartegnar Syndrome

→ ass with Rhinitis & sinusitis b/c

immotile cilia of nose & sinus

→ + dextro-cardia + situs inversus

ex: Young's Syndrome

→ + Dextrocardia + Azospermia "subfertility"

So At Clinic → case of Bronchiectasis, what's importance of  examination?

1 = Rt side  failure b/c Chronic resp disease

→ Hypoxia → Pulm HTN → Cor-pulmonale

2 = Dextrocardia → immotile Syndrome

Acquired

• Children → DTB

▷ pneumonia = Pertussis "whooping cough"
= measles

▷ Foreign body Aspiration

↓
partial Obstruction → foreign body infected
+ local inflam → severe secretion "mucost"

→ difficult to get out b/c foreign body

→ good media for infection + difficult

to Tx → Uni-lat bronchiectasis



Adult

TB

Pneumonia

Bronchial tumor "ex bronchogenic carcinoma"

↓
mass → partial Obstruction → ↑ secretion

→ good media for bact

⊕ Pt with tumor → ↓ immune

Aspergillosis "Complicated B. Asthma"

NOTE

Fungal infection → Aspergillosis

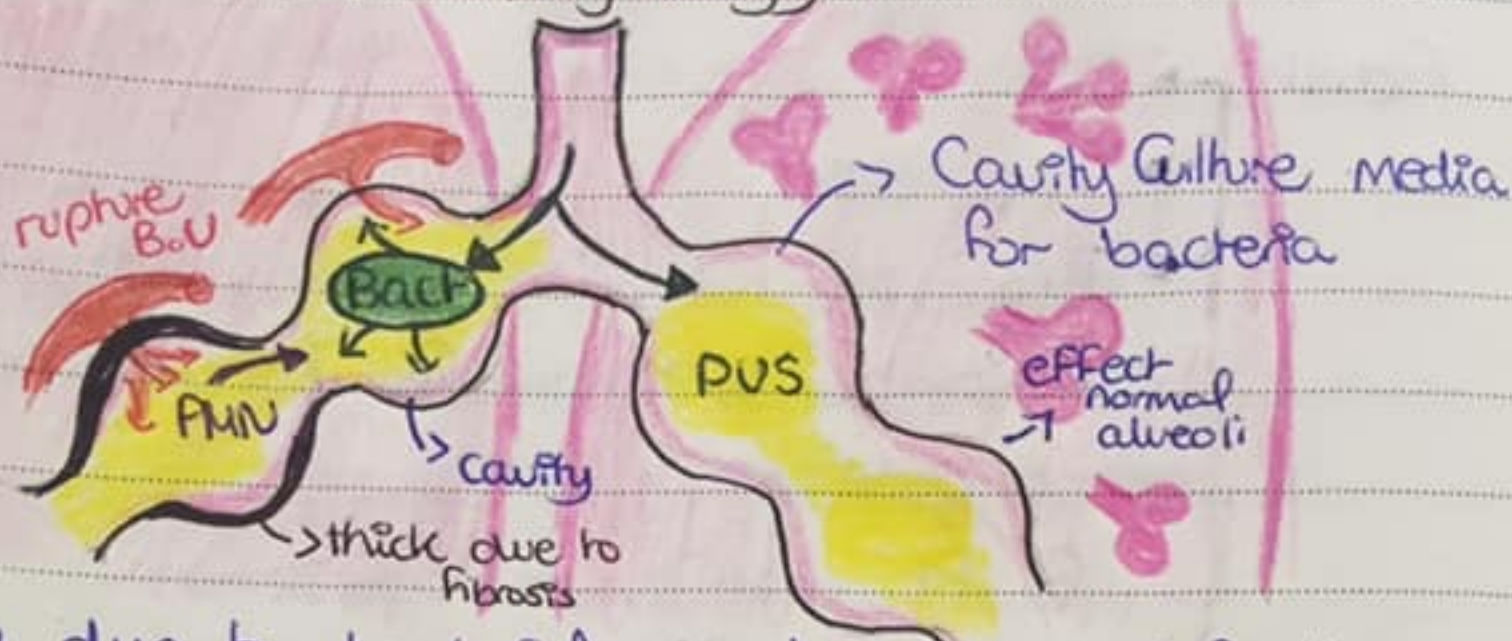
Cause Broncho allergic Aspergillosis or Broncho-pulm
Aspergillosis

1 = ↑ severity of Asthma b/c ↑ sensitivity

→ IF severity ↑ more & complicated Asthma → cause
Bronchiectasis

So most respiratory disease that cause recurrent infection
ex (Asthma + COPD + lung Cancer) cause Bronchiectasis

Pathophysiology



- Cavity due to bacterial enz that cause infection & Destruction → thickening of Cavity by Fibrosis
- Dilatation effect lung alveoli → inflam of neighboring tissue
- Bact eat necrotic tissue → immunological reaction
→ neutrophil PMN try to defense → die & become PUS cell
- with necrotic tissue → PUS
- PUS accumulated in abnormal cavity → multiple Abscess
- Abscess have bact that multiply
- BoU under epthi will be effected → rupture → hemophysis "large amount"
- Bact in cavity → Putrefaction → gases → Bad order = Halitosis
→ DD tonsillitis + pharyngitis

NOTE → How is bronchiectasis one of OAD & the pathophysiology is dilation of airway?

b/c Abscess in Cavity → narrowing of airway

NOTE Suppurative lung disease "PUS"

1 = Bronchiectasis

2 = Lung Abscess on top of pneumonia "Staph. aureus"

3 = emphysema "PUS in Pleura"

& all Cause Clubbing [any chest abscess even 
ex: IE cause Clubbing]

Symp FAHIM $\rightarrow$ low grade fever "Chronic" / headache due to $\rightarrow$ infection $\rightarrow$ cough

- Productive Cough $\rightarrow$ Large amount "Copious" of Purulent sputum

Positional mainly $\rightarrow$ when Pt on one side $\rightarrow$ Abscess

drainage $\rightarrow$ go down to airway $\rightarrow$ irritation to cough receptor $\rightarrow$ large amount of PUS

more in morning, & when tapping on chest by Cupped hand "Tx"

- Dysnea

- wheeze

- Hemoptysis $\rightarrow$ massive amount "up to 500ml"

b/c necrosis of wall $\rightarrow$ rupture of subepith

Bov $\rightarrow$ Bleeding

NOTE "Atypical Presentation"

Some Pt present only with hemoptysis "Pure Blood" without sputum $\Rightarrow$ Dry Bronchiectasis

NOTE

Bronchiectasis with airway effect normal lung C.T and cause pneumonia "surrounding cavity"
→ so at late stage loss of lung architecture

- Pt with bronchiectasis is always a source of infection
How to know if it's superimposed with other infection?

1 = amount of sputum ↑↑

2 = fever high grade

3 = CRP ↑↑

- Complication of Bronchiectasis

1 = Lung Collapse → if secretion completely obstructs airway

2 = emphysema → when cavity open to Pleura ⇒ fistula

3 = Pneumonia + loss of lung architecture

4 = Pneumothorax → when cavity clean "No PUS" after Tx
& Pt has fistula → air in Pleura

↳ MCC of pneumothorax in OAD

1 = emphysema

2 = Bronchiectasis

3 = B. Asthma "rare"

5 = Resp Failure "Type 1 mainly" b/c chronic

Systemic Complication $\rightarrow$ Abscess spread as septic foci in all body

1 = Septicemia

2 = Brain Abscess

3 = Pyelonephritis

4 = renal Abscess - etc

- Hypoxia $\rightarrow$ Pulm HTN + Cor Pulmonale

- 2nd Amyloidosis

$\hookrightarrow$ due to Chronic infection $\rightarrow$ production of para-protein

" Abnormal P₁ "Ab" from plasma cells "AA type"

$\rightarrow$ Large amount $\rightarrow$ Deposit in organs esp kidney

$\rightarrow$ Amyloidosis, nephrotic syndrome & renal failure

* Note AL type $\rightarrow$ Pri Amyloidosis "Abnormal P₁ NOT due to infection"

Sign of Bronchiectasis

• general = FAHM

wt loss esp if TB

= Clubbing "any chest Abscess"

Local • Barrel chest "OAD"

- Could be normal esp if cavity clear "No Abscess or Pt has No Collapse"

so Normal examination doesn't exclude

- MC sign by Auscultation

→ Coarse Creptation in inspiration & expiration

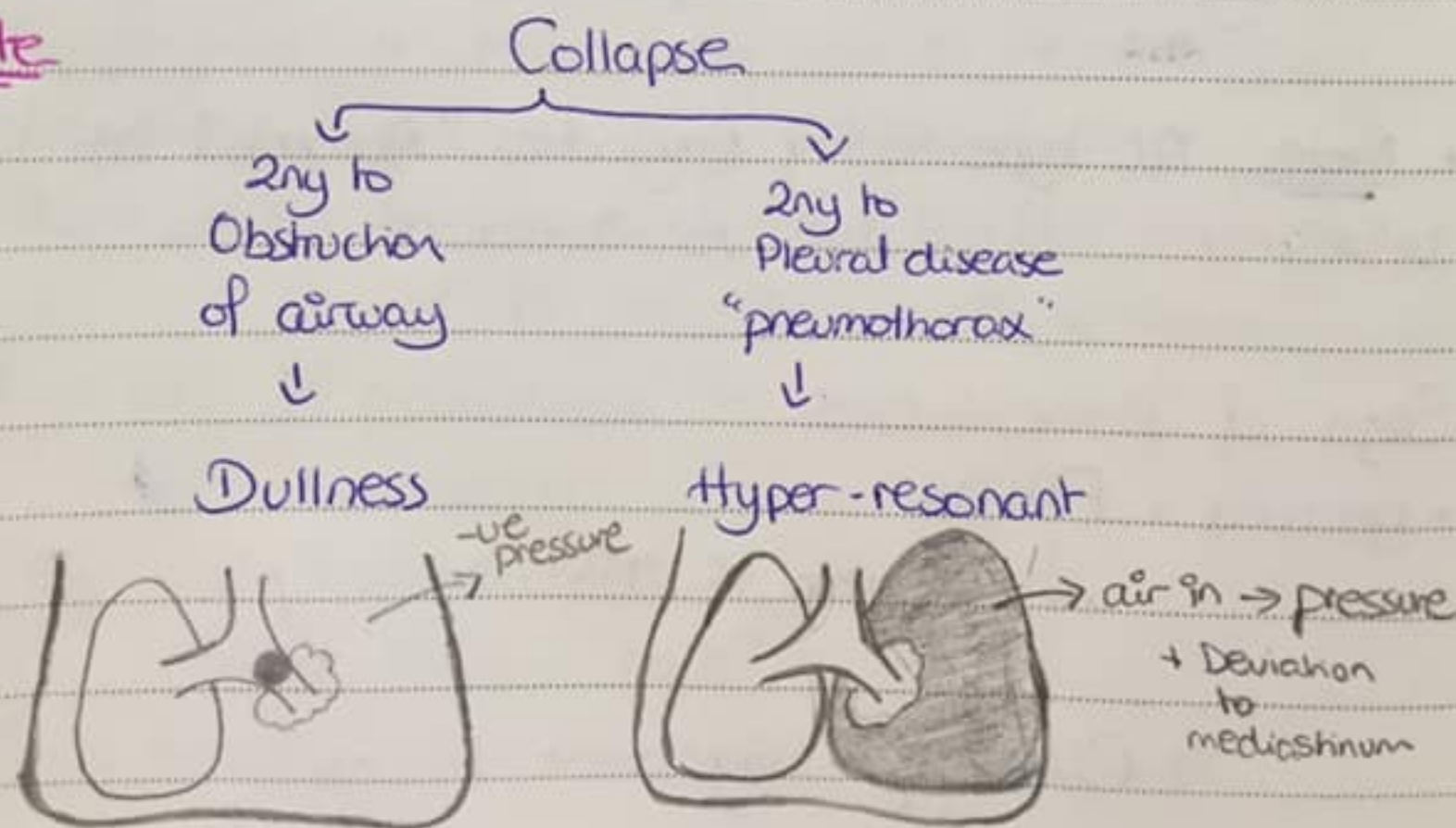
↓ by Cough or mucolytic or Tx "Clean airway"

- evidence of pneumonia "2ny to bronchiectasis"

- Silent Chest if Collapse

& Dullness by percussion "No air" -ve pressure

Note



Inv

PFT $\rightarrow$ FEV₁ < 80% of predictive
FEV₁ / FVC < 70%.



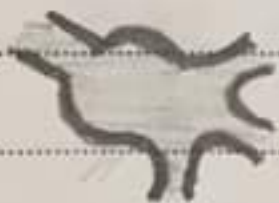
give Salbutamol
No improvement } Not reversible
↓
> 15%
↓

Diff btw it & COPD

by imaging to see Dilatation
& thickening of airway with Abscess

- CXR $\rightarrow$ early normal "so Not exclude"
Late Dx

show dilation & thick airway



$\rightarrow$ Gloved Finger appearance



Tram lines "Thick wall"



$\rightarrow$ Honey-Comb appearance due to cavity lesions

- CT-scan $\rightarrow$ Dx even in early stage



"Honey Comb appearance"

(Best - Gold Standard - Inv of Choice)

Bronchoscopy → used before for Dx
→ know to Tx hemophysis → Close BoV

NOTE Pt with hemophysis doesn't die from hypovolumic shock even though blood is large in amount
BUT die from Clot formation which close airway → suffocation → RF
Tx by resuscitation + bronchoscopy.

ABG → RF type 1

(Inu to detect underlying cause)

1) Saccharin test ⇒

taste test to Dx immotile cilia syndrome

- tab in nostril of nose & wait until Pt taste sugar

b/c cilia in upper airway will move it down

if Pt tasted sugar in < 20 min ⇒ Cilia move normally

if Pt didn't taste sugar → Dx immotile cilia

2) Ig filter ⇒ in case of hypogammaglobulinemia

3) Mantoux test ⇒ in case of TB

Tx $\Rightarrow$ No Tx $\approx$

- **↑ Prognosis** $\rightarrow$ Physiotherapy ONLY
- Postural Changes according to most area that have cavity $\rightarrow$ make cavity upper most so secretion get down & be removed
- by cupped hand $\rightarrow$ freq tapping $\rightarrow$ cause vibration to move secretion "10-20 rapid tap"
- while doing it tell Pt to take deep breathing rapidly & freq times to help move secretion
- $\rightarrow$ Stim Cough receptors $\rightarrow$ Cough $\rightarrow$ Clearance airway
- Collect sputum for Culture to know organism
- $\rightarrow$ so can give Ab "ex: AFS for TB"
- B/c recurrent secretion $\rightarrow$ Physiotherapy done twice daily (5-10 min)

• **Tx for Symp** "↓ inflam & secretion"

1 = Bronchodilator = SABA

2 = ICS

3 = mucolytic = DNase 2.5mg inhaler

$\hookrightarrow$ liquification of secretion

4 = Ab if Supper opposed by infection

by $\uparrow$ fever - $\uparrow$ sputum - $\uparrow$ CRP - leukocytosis

- Subject:
- **Surgery** → Lobectomy if uni-lateral "ex: foreign body"
so young pt with focal bronchi effected remove it
before it spread to other lung

DD of chest scar due to lung surgery

- emphysema
- Bronchiectasis
- Hydatid cyst
- Lung Cancer "rare" b/c mainly present
Late stage - mets"

NOTE

Yellow nail syndrome "Congenital"

Yellow nail

Scoliosis

Bronchiectasis

NOTE

Rheumatoid Arthritis + Sjogren syndrome ass
with bronchiectasis.

"Cystic Fibrosis"

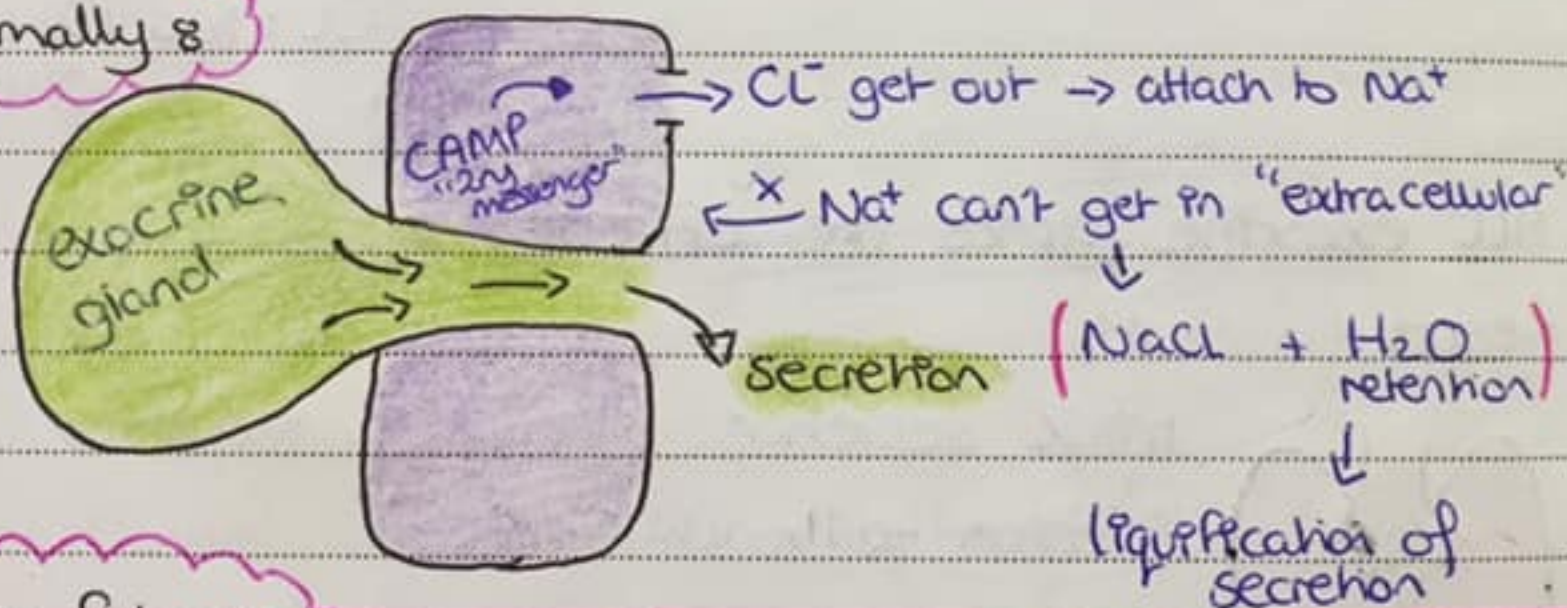
AR disease

Chromosome 7 $\rightarrow$ long arm $\rightarrow$ Position 508 $\rightarrow$

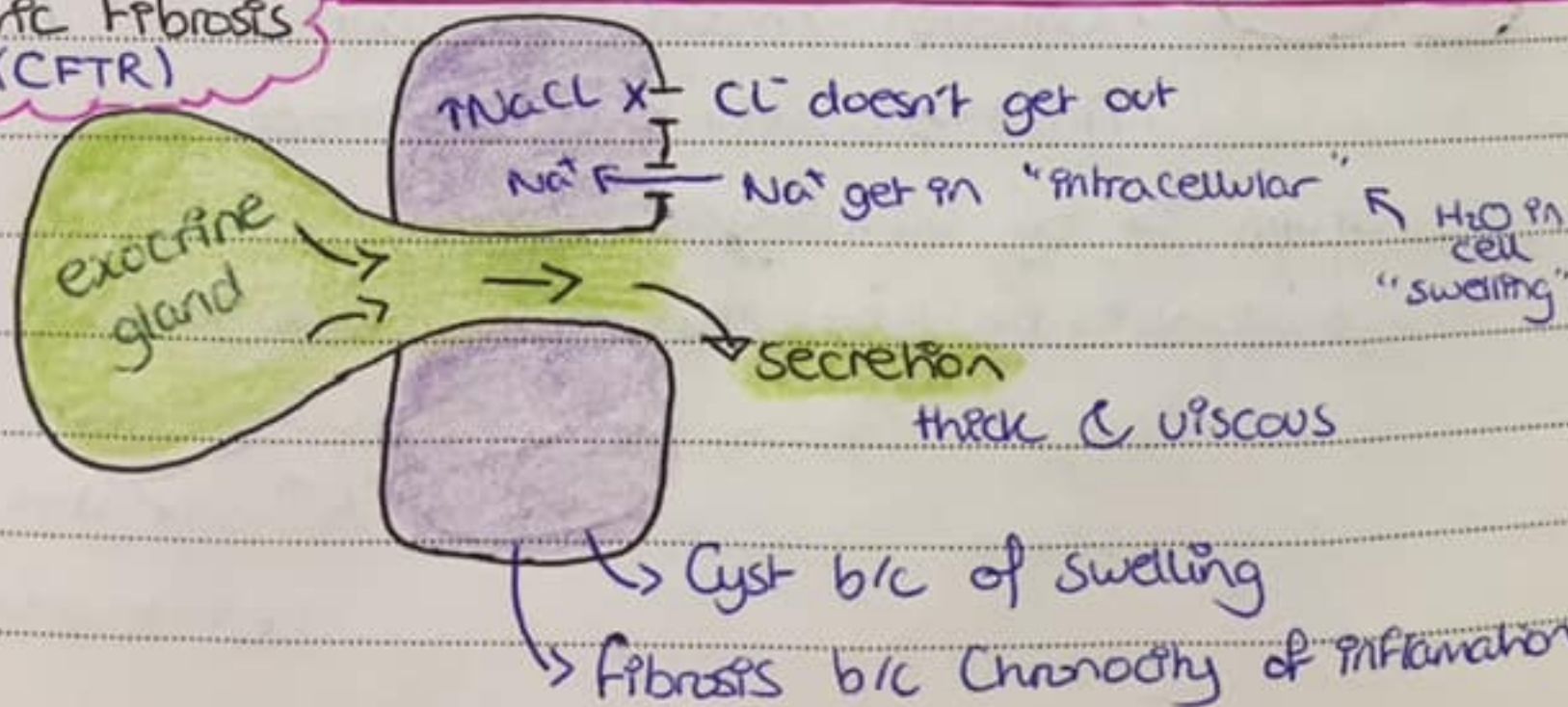
Trans-membrane conducting regulator prt $\rightarrow$ regulate permeability of cell membrane

- in Cystic Fibrosis $\rightarrow$ Deletion $\rightarrow$ Abnormal permeability
- most fatal hereditary disorder $\rightarrow$ esp in Caucasians.
- MCC of Death $\rightarrow$ respiratory failure

Normally



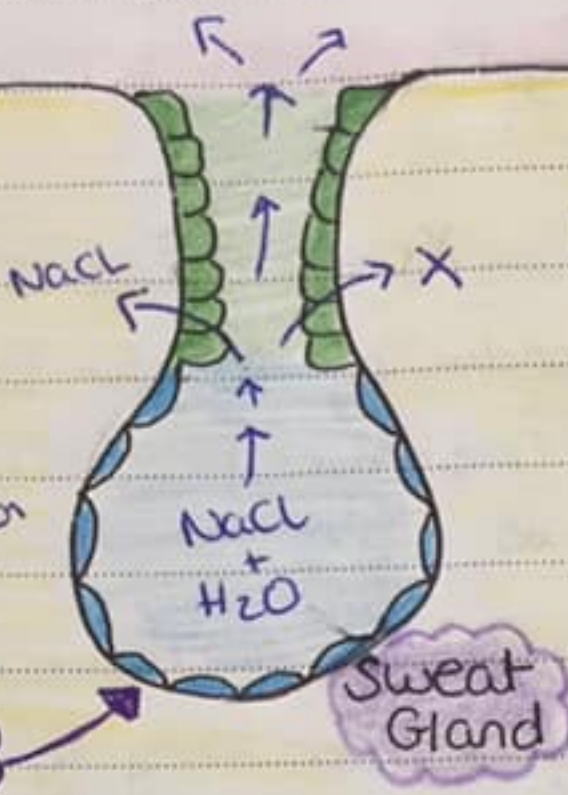
Cystic Fibrosis (CFTR)



Normally

Ductal cell reabsorb NaCl
 ↓
 Sweat have low concentration of NaCl

Symp



Cystic Fibrosis

No reabsorption
 ↓
 Sweat high concentration of NaCl
>60 mmol



Dx of CF

by sweat test

(NaCl >60 mmol)

(Cl:Na 2:1 or 3:1)

All exocrine glands are effected &

Resp:



thick secretion will cause recurrent infection → Bronchiectasis

- Children → mainly Staph. aureus
- Adult → pseudomonas aeruginosa

Staph Tx by vancomycin

pseudomonas by Anti-pseudomonas → Ciprofloxacin
 → Ceftazidime

"4th generation

Cephalsporins

• Resp failure $\rightarrow$ cause of Death at 40s

in UK upto 60s b/c prt gene inhaler therapy

• Sinusitis

• recurrent nasal polyps $\rightarrow$ DD B. Asthma "Churg-Strauss Syndrome"

Cystic Fibrosis



• Chronic pancreatitis due to thick secretion that close pancreatic duct

$\downarrow$ elastase enzyme in stool

• Chronic pancreatitis $\rightarrow$ Fibrosis $\rightarrow$ B-cell not working $\rightarrow$ DM

So [Bronchiectasis + DM $\rightarrow$ Cystic Fibrosis
emphysema + Liver Cirrhosis $\rightarrow$ α_1 anti-trypsin]

GIT • meconium plug in pediatrics

• Constipation + malabsorption

• Steatorrhea + AKED Def due to $\downarrow$ Bile excretion
esp vit D $\rightarrow$ osteomalacia osteoporosis - rickets

osteoarthritis

So Skeletal Abnormality very common with CF

• Obstruction of bile duct $\rightarrow$ jaundice + Liver fibrosis

- Genetourinary $\rightarrow$ sterility upto 95% or more
b/c sperm can't get out from vas
deferens b/c thick secretion $\rightarrow$ Close

Note Sub Fertility

Bronchiectasis + Sterility $\rightarrow$ Cystic Fibrosis

Bronchiectasis + Azoospermia $\rightarrow$ Karynagar + Young's

Rectal prolapse $\rightarrow$ DD Cystic Fibrosis
in pediatric Celiac disease

Failure to thrive & mental retardation in Children

Resp problem mainly in Adults

Inv sweat test "Confirm Dx"

PFT $\rightarrow$ Obstruction pattern of Bronchiectasis

$\downarrow$ elastase in stool due to Chronic

Tx Supportive Tx

- Tx Bronchiectasis
- Sterility $\rightarrow$ IVF
- vit supply
- Bile salt ursodeoxycholic acid
- Ca^{+} + bisphosphonate $\rightarrow$ for osteoporosis

NOTE

you can Dx CF before symp by genetic study screening

Triple Drg regimen $\rightarrow$ improve prognosis (2Ab + DNase)

1) Inhalor Ab

'Tobramycin "Aminoglycoside daily"

2) Azithromycin "macrolid" wkly

3 Tab coarse Day by Day

& Ab monthly to cover infection

3) DNase "mucolytic inhaler"

b/c Cause of Death is RF from bronchiectasis due to recurrent infection $\rightarrow$ prevent it by Dx CF early by

1= Family screening for any pt with CF

2= Intra uterine by Amniotic Fluid genetic study

3= After Delivery $\rightarrow$ Blood test 'Aminoacids $\downarrow$ in CF'

ILD

more than 200 disease that share the same symp & signs



replacement of normal CoT by fibrosis



slowly progressive so gradual symp

PFT - ABG - CXR → early normal b/c →

HRCT scan → Better for Dx

if -ve Not exclude

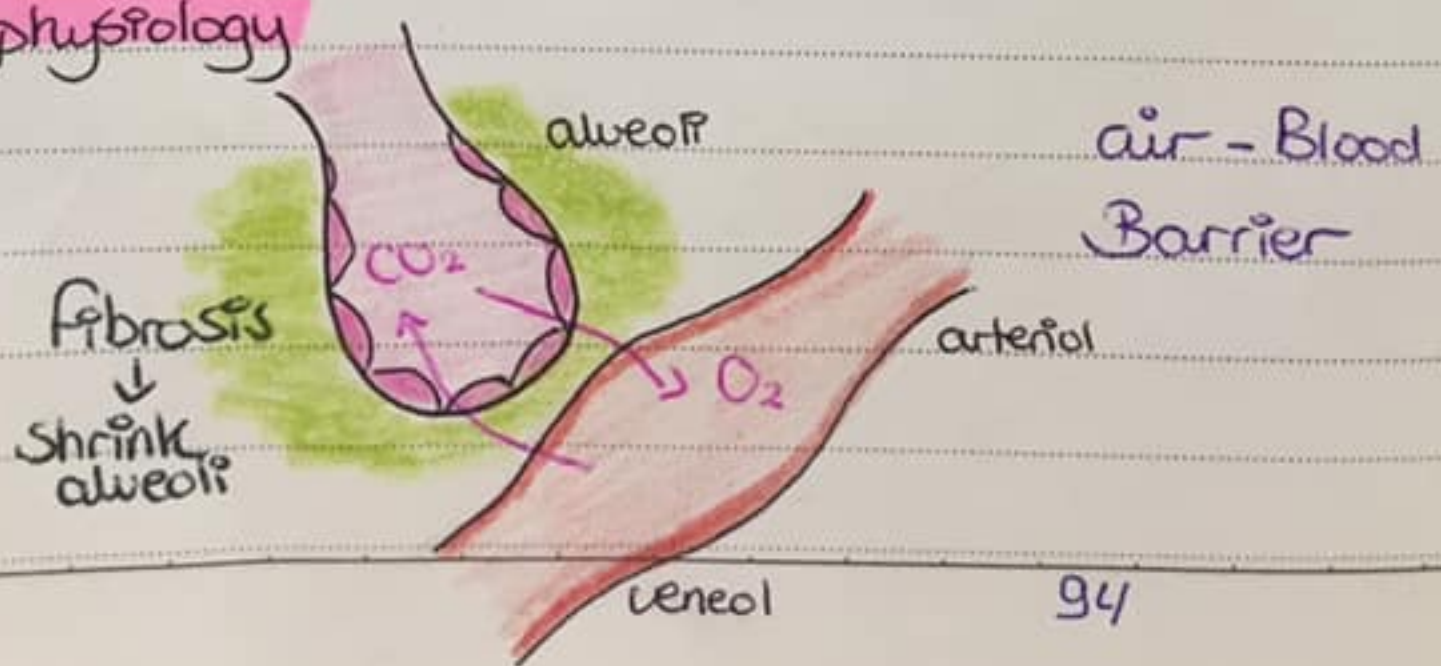


Lung Biopsy "GOLD standard"

by bronchoscopy if lesion central

Percutaneous if lesion peripheral

Pathophysiology



Pathology in interstitial $\rightarrow$ fibrosis $\rightarrow$ slowly progressive
& alveoli shrink

1 = Thickness $\rightarrow$ Limitation of expansion of alveoli

$\rightarrow$ can't inflate $\rightarrow$ **Dysnea** "Presenting symp"

gradual + slowly progressive "from stage 1 $\rightarrow$ 4"

"MC presenting symp b/c cough receptors in airway that are normal at early stage "OAD cuts"

NOTE

some cases have rapid onset of fibrosis $\rightarrow$ present with acute severe Dysnea worsening rapidly "less common"

2 = After Dysnea $\rightarrow$ Chronic inflammation will cause irritation of cough receptor in airway

Dry Cough b/c less goblet cell in lower airway!

3 = Pt never have **wheeze** b/c airway normal "Problem in interstitium" $\rightarrow$ so wheeze exclude ILD

4 = **Fine Crpitation** b/c fibrosis cause shrinking in alveoli
 $\rightarrow$ during inspiration $\rightarrow$ re-opening of collapsed alveoli
"Most imp sign" / Not $\downarrow$ by cough (cough $\rightarrow$ less)
end inspiratory Crpitation $\approx$ Fibrosis

NOTE Case $\rightarrow$ Pt working in a factory for 20yrs
Presented with Dysnea for 6m - slowly progressive
Started on exertion & know with minimal activity
 $\rightarrow$ Think ILD

INX

X-ray $\rightarrow$ white b/c $\rightarrow$ $\downarrow$ air "alveoli smaller"
 $\downarrow$ $\rightarrow$ $\uparrow$ Fibrosis "solid"

reticular shadow "network like" + nodules "dots"
= reticular nodular shadow $\Rightarrow$ indicate fibrosis

ABG

Hypoxia $\rightarrow$ b/c O_2 can't get in to blood b/c thickness $\uparrow$
& Permeability $\downarrow$ $\rightarrow$ Hypoxia up to < 60 mmHg
But CO_2 rarely effected b/c more permable $\rightarrow$ can get
out so not elevated

$\Downarrow$
(RF type 1) most common

end stage ILD Fibrosis & thickness $\uparrow$ more than
effect even $CO_2 \rightarrow \uparrow$ in blood
"RF type 2" "rare"

- So
- main type of RF in ILD is Type 1
 - RF type 2 may occur in ILD

$O_2 \downarrow \rightarrow$ V/Q of Pulm BoV $\rightarrow$ Pulm HTN $\rightarrow$ Cor pulmonal

PFT $\rightarrow$ FEV₁ < 80% of predictive
FEV₁ / FVC = > 70%

DLCO $\rightarrow$ $\downarrow$ b/c thickness of diffusion $\uparrow$

NOTE

1st inv change in ILD is DLCO

other inv ex (PFT - CXR - ABG) early are normal

So

Best parameter of PFT in B. Asthma = PEFM

Best parameter of PFT in COPD = FEV₁

Best parameter in ILD = DLCO

Comp RF Type 1 $\rightarrow$ MCC of Death

Rt $\heartsuit$ Failure

C/P Symp $\Rightarrow$ Dysnea - Dry Cough - No wheeze

Sign

I = small chest "b/c $\downarrow$ volume of lung"

P = Chest expansion $\downarrow$ Bi-lateral (esp base) < 3cm

P = resonant $\rightarrow$ until dense fibrosis "dull late stage"

A = $\downarrow$ air entry - vesicular breathing

Fine Crackles
"Best"

DD of fine Crispitation at base of lung = LF $\rightarrow$ Failure
How to diff?

Fine Crispitation $\downarrow$ by leaning forward in ILD
& Not in HF

NOTE Silent PFT = Silent deterioration of lung function
PFT result normal although function of lung $\downarrow$

Causes of ILD

★ Known Causes ★

1) Pneumoconiosis

Occupational lung disease 'Asbestosis - Silicosis - Berylliosis'

2) Extrinsic allergic alveolitis (EAA)

= hypersensitivity pneumonitis = farmer lung

due to exposure to ~~some~~ some fungal spore or avian prt

or wheat or barley Ag for a long period $\rightarrow$ cause

Inflammation Chronically $\rightarrow$ Fibrosis

3) Radiation

radiotherapy or somatoform disorder

4) Drugs

- GOLD - D-penicillamine] cause also nephrotic
- methotrexate - Amiodarone] cause lung + Liver Fibrosis
- Cephalosporine - Nitrofurantoin] Ab
- Bleomycin (Chemotherapy)

NOTE

- If Pt Stop drug $\rightarrow$ improved
- Methotrexate on long term cause pneumonitis "acute on top of Chronic "emergency"

5) ARDS

6) Aspiration pneumonia "could be uni-lateral - Rt side mainly lower lobe"

★ Unknown Cause ★

1) IPF idiopathic Pulmonary Fibrosis "MCC" 85%
= Cryptogenic Fibrosing alveolitis

2) Sarcoidosis "non-caseating granulomas"

3) Autoimmune disease

A) CTD $\rightarrow$ SLE

Sjogren syndrome

Scleroderma

RA

} joint pain + skin rash

B) IBD $\rightarrow$ CD - UC

$\rightarrow$ non-caseating granulomas

C) Vasculitis $\rightarrow$ Wegner's granulomatosis
 $\rightarrow$ Goodpasture's Syndrome $\left\{ \begin{array}{l} \text{lung} = \text{hemoptysis} \\ \text{kidney} = \text{hematuria} \end{array} \right.$

4) Histiocytosis X

Idiopathic eosinophilic granuloma

NOTE

- Non caseating granuloma $\rightarrow$ commonly cause ILD
- Caseating granuloma $\rightarrow$ TB $\rightarrow$ commonly cause Bronchiectasis

Hx of ILD "3 main Q"

1 = Occupational Hx

2 = Drug Hx - Radiation

3 = Rheumatological Hx $\Rightarrow$ Skin rash - joint pain

NOTE

most of ILD effect Basilar Bi-lateral $\downarrow$ IPF Asbestosis } commonly have Clubbing



except $\rightarrow$ 1 = Sarcoidosis
2 = Silicosis
3 = TB "Post Pry" reactive
4 = Ankylosing Spondylitis
5 = radiation
6 = EAA

ILD don't have Clubbing except
(IPF - Asbestosis) commonly

* Sarcoidosis cause uni-digital Clubbing rarely!

↳ HCO

Sarcoidosis rarely cause Clubbing (✓)

Sarcoidosis commonly cause Clubbing (x)

Clubbing is a feature of Sarcoidosis (x)

Examination imp in general to see Clubbing!

BAL

"Broncho-alveolar lavage

by using Fibroptic Bronchoscope → with Normal Saline
irrigation & Aspiration to see cells in airway

"Cells are in paracyma but can't get to it so
we use BAL to take them from airway"

- IPF ⇒ neutrophil + eosinophil
 - Sarcoidosis ⇒ CD4 T-lymp
 - EAA ⇒ CD8 T-lymp
- } Not specific but help Dx
(Dx test = lung biopsy)

CBC $\rightarrow$ anemia of Chronic illness

Lymphopenia b/c $\uparrow$ in airway "esp sarcoidosis"

neutrophil $\uparrow$ in EAA

NOTE

EAA = Allergic you will think eosinophilia $\exists$
but it's neutrophilia

Rheumatoid factor $\Rightarrow$ +ve in most ILD

ANA $\Rightarrow$ +ve in most ILD

NOTE MC granulomatous disease lead to lung fibrosis
is Sarcoidosis

Idiopathic Pulmonary Fibrosis IPF

(Unknown Cause)

$\left[\begin{array}{l} \sigma^+ > \text{♀} \\ > 50 \text{ yrs} \\ \uparrow \text{ smoking} \end{array} \right]$

T-Lymph abnormal activation $\rightarrow$ Stim
Fibroblast $\rightarrow$ Fibrosis basal

• Abnormal reaction start at airway $\rightarrow$ Chemotaxis

1st Fibroblast $\rightarrow$ Fibrosis

then inflammatory cell precipitate (neutrophil + eosinophil)

So in BAL

1 = T-lymph = Alarm sign that fibrosis will start & severe
→ indicate that process is starting again

& Steroid have a role → to prevent Alveolar activity

2 = neutrophil + eosinophil "Not T-lymph" فان القطار

→ Fibrosis already established → No benefit of using Steroid, Pt will have S/E of it only!

C/P FAHM + Dysnea + Dry Cough
Sign → Fine Crispitation + Clubbing

INV. CBC Anemia

• ESR - CRP ↑ → DD infection / inflam / malignancy

• ↑ LDH → indicate activity of disease "start Fibrosis"

DD hemolytic anemia - Bone disease
hypothyroidism

• ABG → Type 1 RF

• PFT → > 70%

• CXR → early = normal

late = reticulonodular shadow

Honey Comb appearance → DD ILD

• HRCT → early

↓

DIFF by HRCT

Branchiactasis

• Activity of IPF "relapse" by 8

1 = T-lymp in BAL

2 = LDH ↑ - CRP ↑ - ESR ↑

Dx

Hx - Ex - Inv

inv if +ve No need for Biopsy

if -ve or in doubt → do Biopsy

NOTE

RF +ve 10%

ANA +ve 20-30%

Tx • Steroid ⇒ if T-lymp is pre-dominant

0.5 mg / kg daily

• Azathioprine

2-3 mg daily

• morphine improve dysnea

NOT improve
prognosis

• No benefit for lung transplant "not effective -
old age - unoperable"

Prognosis

Poor prognosis → survival rate 3yrs of Dx

rare live >5yrs

★ **NOTE** Drugs under trial "some cases Delayed Fibrosis"
warfarin - NAC - Interferon gamma

Note Acute on top of Chronic "rare"

"Hamman-Rich syndrome"

very poor prognosis "survival rate 3m"

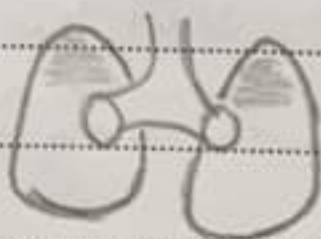
Sarcoidosis

♀ > ♂
young
smoking protective
more in Black

Non caseating granulomas

multi-systemic disease

mainly effect resp system



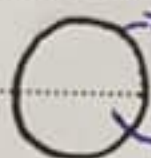
MC presenting sign (BHL) 90%

"Bi-lateral hilar lymphadenopathy"

⊕ Lung Fibrosis "Apical" 20%

multi-Systemic

▶ granulomas ⇒



Fibrosis

Inflam cells →

T-lymp
Plasma cell
macrophage
giant cell
Fibroblast

Pathophysiology

CD4 - T-lymp

"Delayed hypersensitivity type 4"



Abnormal activation



Precipitate in Airway

BHL



then infiltrate lung "Fibrosis"

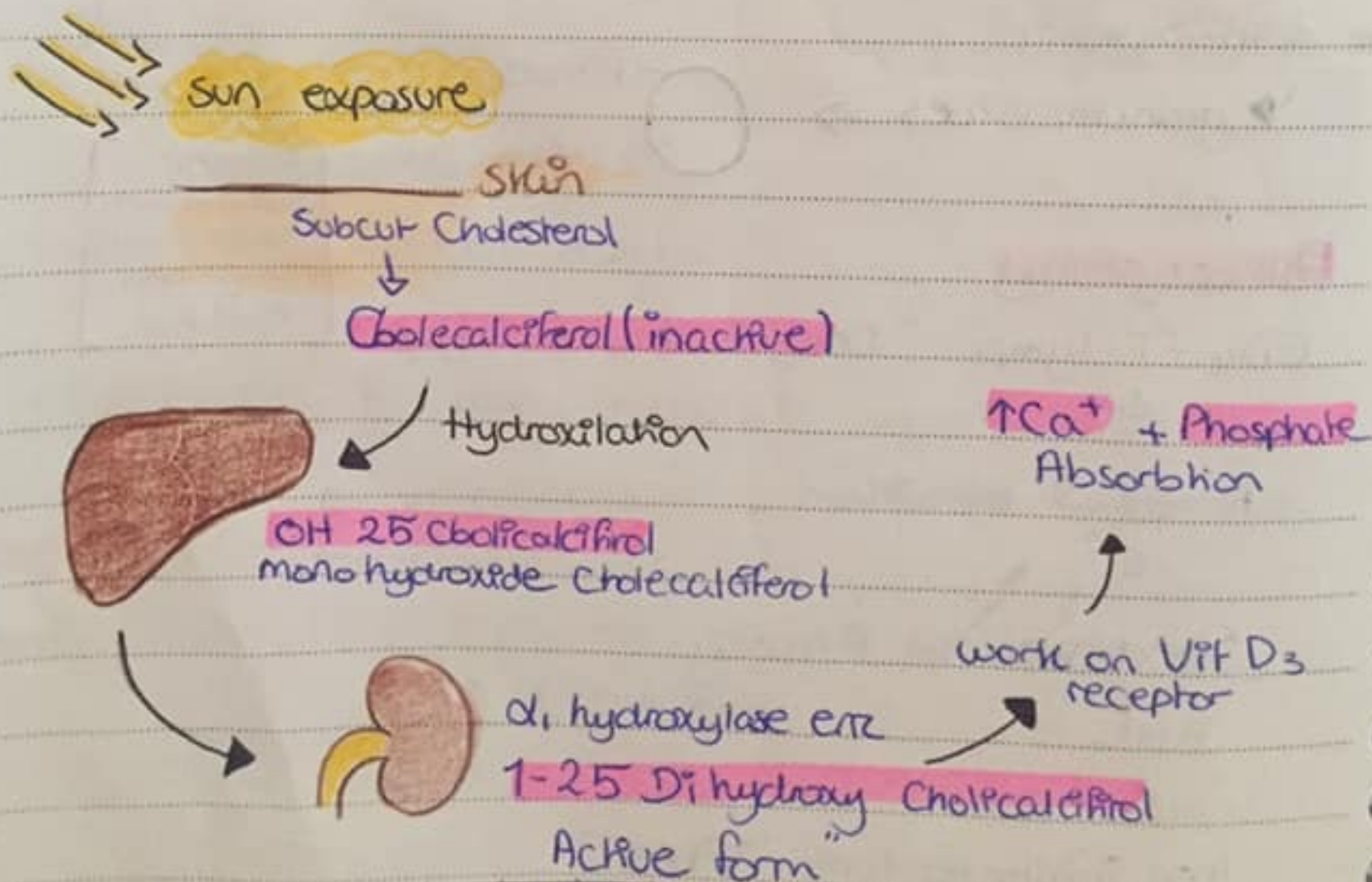
NOTE Pt with sarcoidosis $\rightarrow$ always Mantoux test is falsely -ve

bic defect in CD4 "low in Blood
& work Abnormally!"
 $\uparrow$ in airway"

MCQ All of following are compatible with each other

- Pneumonia + Strepto-pneumoni
- Klebsila pneumonia + Alcohol
- TB + Bronchiectasis
- Sarcoidosis + -ve tuberculin skin test
- COPD + smoking

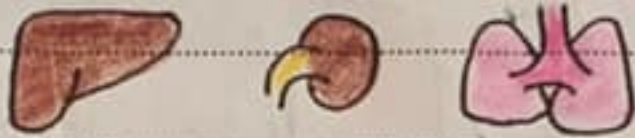
Vit D Activation



How to diff btw VitD + Parathyroid?

$\uparrow \text{Ca}^{2+}$ $\uparrow \text{Ca}^{2+}$
 $\uparrow \text{Phosphate}$ only & $\downarrow \text{Phosphate}$

Sarcoidosis $\rightarrow$ in lung a. Hydroxylase like activity
so Vit D₃ in Sarcoidosis activated in 3 organs

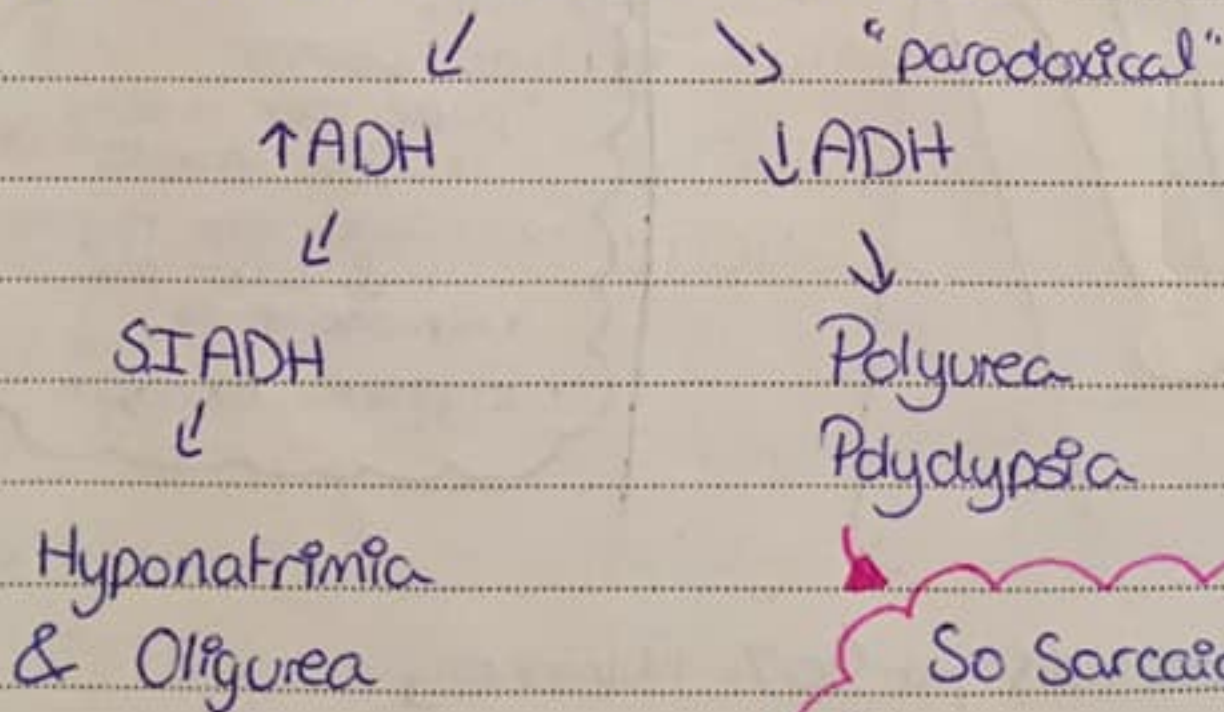


$\uparrow \text{Vit D} \rightarrow \uparrow \text{work} \rightarrow \uparrow \text{Ca}^{2+} + \uparrow \text{Phosphate}$

Hypercalcemia $\rightarrow$ Polyurea "osmotic diuresis"
+ Polydipsia

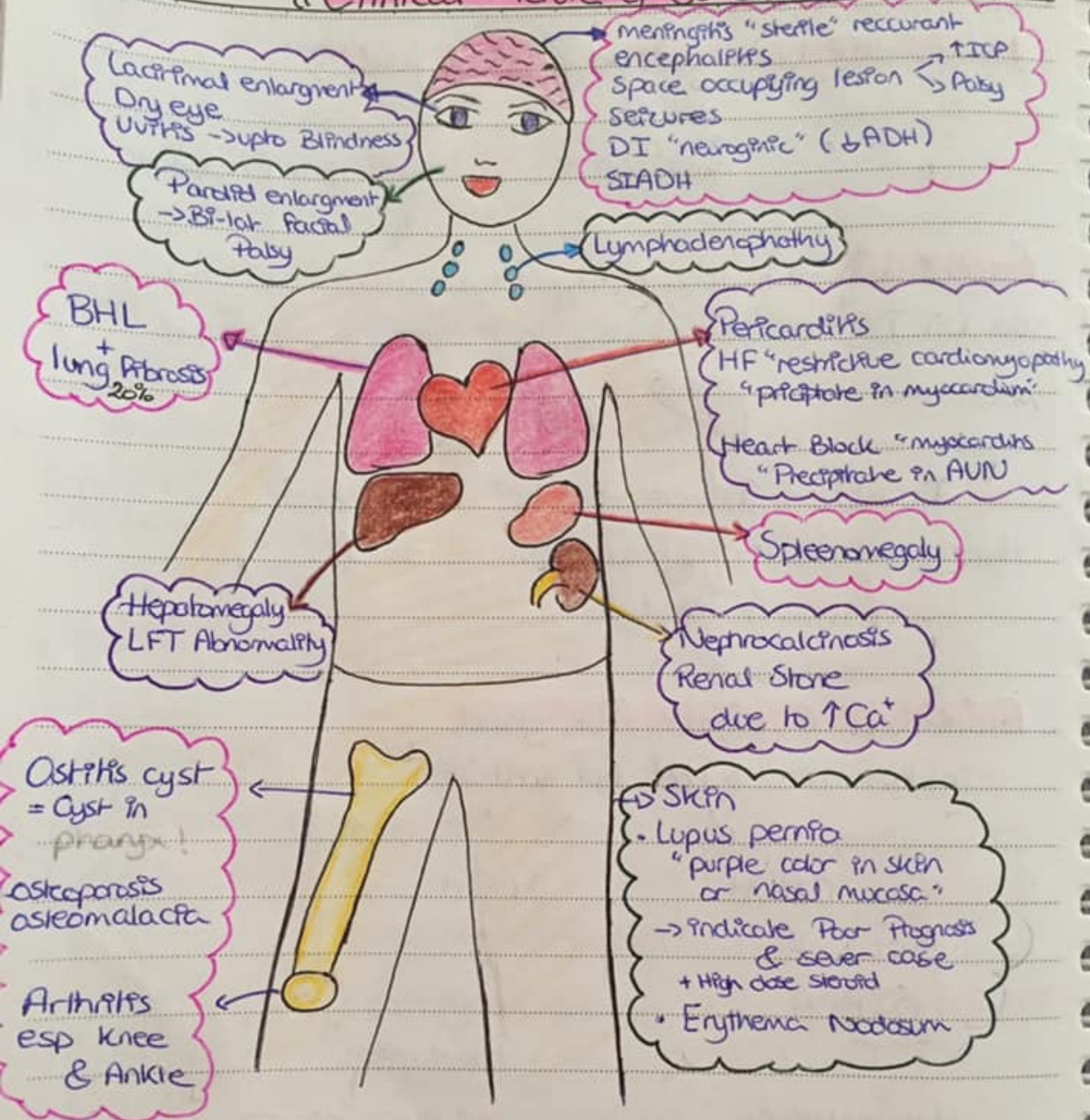
Granuloma if effect Pit gland

Inflammation $\rightarrow$ Post Pit irritation



So Sarcoidosis cause
Polyurea by 2 ways $\Rightarrow$
($\uparrow \text{Ca}^{2+}$ - $\downarrow \text{ADH}$)

Clinical Picture of Sarcoidosis



C/P of Sarcoidosis → 90% Pulmonary
 → 10% extra-Pulmonary

40% Insidious onset

DD of BHL $\Rightarrow$ 1 = Sarcoidosis *

2 = Infection $\rightarrow$ TB* - mycoplasma

3 = Cancer $\rightarrow$ Lymphoma* - Lung Cancer

4 = Sarcosis

5 = EAA

DD of hepatosplenomegaly with lymphadenopathy

1 = Sarcoidosis

2 = TB

3 = HIV

4 = Toxoplasmosis --- etc

Granulomatous disease of Liver

1 = Sarcoidosis

2 = Pyl Biliary Cirrhosis

3 = Schistosoma

INV

CBC = Lymphopenia

PPD test = -ve even if Pt is TB

Ca^{++} = normal or $\uparrow$ "indicate severity"

ACE = Activity $\uparrow$ in 60% of cases & $\uparrow$ in Blood

if $\uparrow$ in CSF indicate CNS involvement

& it's 1st sign of neurological involvement

so ACE NOT for Dx but for monitoring

Case Pt came with fits, How can you know if it was sarcoidosis or other cause ex: electrolyte disturbance?

by CSF sample $\rightarrow$ ACE "very sensitive"

DD of $\uparrow$ ACE in Blood?

- Sarcoidosis
 - TB
 - Silicosis
 - Hyperthyroidism
- So Not specific NOT for Dx $\Rightarrow$ But Sarcoidosis only $\uparrow$ ACE in CSF

- CXR - HRCT = mainly Dx disease

- PFT = restrictive

- ABG = RF Type 1

- Fibro-optic bronchoscope

= Cobble stone Appearance $\rightarrow$ DD in GIT "Cron's" indicate granuloma

- BAL = CD4 lymphocyte

- Biopsy = Best test

Note

severity of disease indicated by 1 = lupus pernio

2 = $\uparrow$ Ca²⁺

Stages according to Imaging technique "CXR - CT"

Stage 1 $\Rightarrow$ BHL only

Stage 2 $\Rightarrow$ BHL + Parachymal infiltration "Not Fibrosis"

Stage 3 $\Rightarrow$ Parachymal infiltration Only] $\downarrow$ regression of

Stage 4 $\Rightarrow$ Dense Pulm Fibrosis] LN size

Tx according to stage

Stage 1 $\Rightarrow$ Self limiting "No Tx"

Stage 2 $\Rightarrow$ (Tx if sever symp) $\begin{cases} \rightarrow 60\% \text{ Don't need Tx} \\ \rightarrow 40\% \text{ need steroid} \end{cases}$

Stage 3 $\Rightarrow$] even if $\rightarrow$ Tx by systemic steroid

Stage 4 $\Rightarrow$] Asymp

Indication of steroid

1 = Stage 3 + 4

2 = Stage 2 if sever

3 = Lupus pernio

4 = Extra-Pulm $\Rightarrow$ CNS - $\heartsuit$ - kidney - eye

5 = Hypercalcemia

NOTE

• Liver involvement is NOT indication for steroid

• ex Löfgren's syndrome $\Rightarrow$ acute stage 1 "BHL + Abnormal Liver function $\rightarrow$ No steroid!"

Tx

Prednisolone 3-4mg/day "1st line"

↓

if Not improved

↓

Immune Supp "MTx + Azathioprine" 2nd Line

NOTE

if you want to give steroid with high dose & prevent cushing S/E

1) give systemic "lower dose" + ICS "↓ systemic S/E"

or 2) give Azathioprine "Steroid sparing Drug" → ↓ dose of steroid
"used even for withdrawal"

Poor Prognosis Feature of Sarcoidosis

1 = Old age

2 = Symp > 6m & Not respond to Tx

3 = Lupus pernio

4 = Stage 3+4

5 = CNS - eye involvement

Mortality rate

1-5% Die from Sarcoidosis

Lung Cancer

MCC of Death = IHD

MCC of Cancer Death = Lung Cancer

mortality from Cancer

♂

- 1 Lung Cancer
- 2 Prostate Ca
- 3 CRC
- 4 Bladder Ca

♀

- 1 Lung Cancer
- 2 Breast Ca
- 3 CRC
- 4 ovarian Ca

- most cases present late "Not operable" Tx by Chemo or radio
- 1,300,000 case Dx yearly
& 95% Die within the 1st yr
- Lung Cancer Death present 18% of Death rate

Risk Factor

1 = Smoking "MCC" = 90% of Cancer are smokers
(↑ 30 times) → Sq. cell Carcinoma "metaplasia"

"Lung Cancer on top of smoking poorest prognosis than non-smoker Cancer"

- ex-smoker - ↓ risk but not back to normal

2 = Silica exposure - Asbestosis "↑ 5 times"

↳ Adeno-carcinoma mainly

3> Air pollution $\Rightarrow$ Cadmium - Chromium

$\hookrightarrow$ Adeno-carcinoma "mainly"

NOTE

- most common type of lung Ca is sq. cell b/c on top of smoking MCC
- most common type of lung Ca in ♀ = Adenocarcinoma
- most common preventable Cancer = Lung Cancer by STOPPING Smoking =
- Passive smoker present 5% of lung Cancer.
- Lung Cancer have a genetic role.

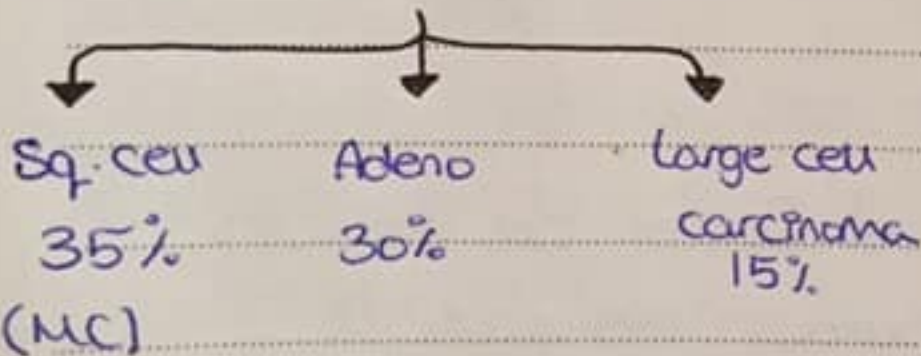
Classification

Non-small cell

Lung Cancer 80%
(NSCLC)

Small cell

Lung Cancer 20%



$\downarrow$
b/c bigger in size may present locally $\rightarrow$ Operable
if mets $\rightarrow$ radio - Chemo

Oat cell
"feature of sarcoma"
 $\downarrow$
go to blood easily
 $\downarrow$
mets rapidly

$\downarrow$
Poor Prognosis
"In-operable"
Tx by radio-Chemo

NOTE

- Adenocarcinoma usually radio sensitive resistance
so mainly Tx Chemotherapy
b/c Adeno is slowly divided & radio effect rapidly
divided cells b/c effect DNA "mutation"
- Sq. cell carcinoma divid rapidly $\Rightarrow$ radio sensitive
- That's why S/E of radiotherapy $\Rightarrow$ mouth Ulcer b/c
mucos memb rapidly dividing cells.
- MTx $\rightarrow$ work like radiotherapy $\Rightarrow$ S/E mouth Ulcer
 $\rightarrow$ effect folate
- MC type $\Rightarrow$ Sq. cell & Best prognosis b/c respond to Tx

Sites

Peripheral

1 = Adeno

Non-Cavity

2 = large cell
Cavitary

Biopsy percutaneous

Central

1 = Sq. cell

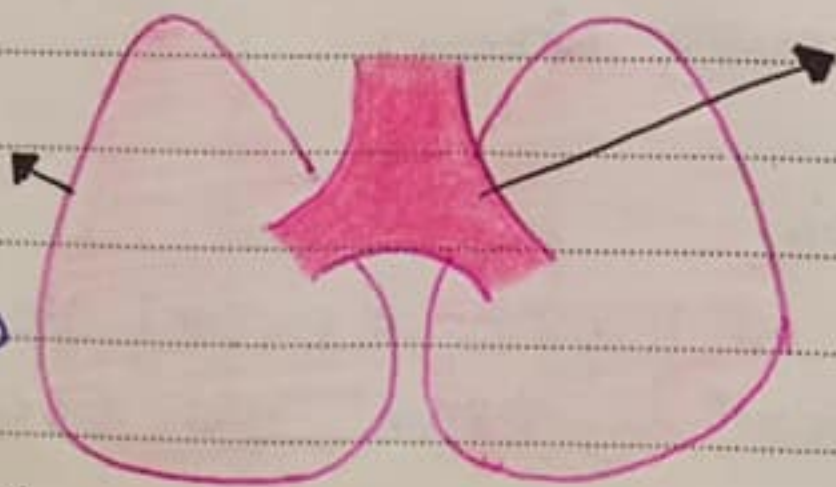
ulcer $\uparrow$ + cavity

"hemoptysis"

2 = oat cell

Non-ulcer / Non-cavity
less hemoptysis

Biopsy Fibro-optic
transbronchial



[CIP any old Pt with S&S of anemia - fatigue]
- Sleepy - wt loss & anorexia - recurrent infection
all are alarm sign $\rightarrow$ think of malignancy.

Resp CIP

1 = Dry Cough "MC symp" 80%

$\rightarrow$ Productive if Blood - infection due to immune

- Cough due to irritation of airway
- Change in pattern of Cough for months.
- mainly dry Cough unless $\Rightarrow$ Ch. bronchitis

2 = Hemoptysis "70%"

b/c cavity $\rightarrow$ rupture BoV in submucosa $\Rightarrow$ massive bleeding

3 = Dysnea 60%

$\hookrightarrow$ mass narrowing airway

4 = Chest pain 40%

peripheral due to invasion of parietal pleura

or pleura effected by inflam "even if mass central"

"less common"

NOTE

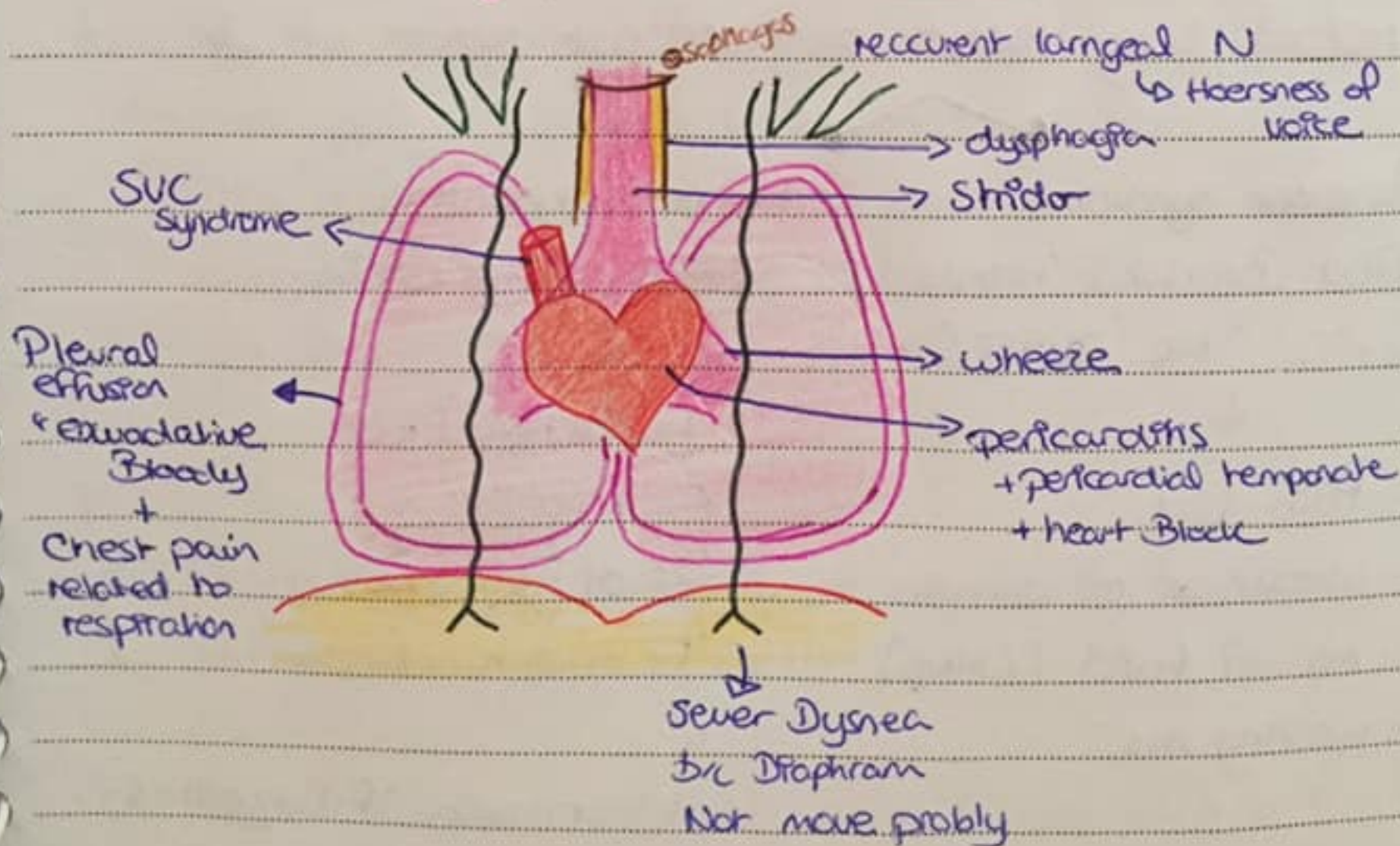
- Cough + Fever + Chest pain $\Rightarrow$ Think pneumonia
- Cough + hemoptysis + pain $\Rightarrow$ Think Cancer
- unresolved pneumonia
= recurrent pneumonia
= refractory pneumonia
= resistance pneumonia

Think Cancer
or TB



(b/c mass cause stagnation of mucus $\Rightarrow$ Ab doesn't reach Bact which lead to bronchiectasis)

Symp of Local invasion



NOTE

SVC Syndrome $\Rightarrow$ Obstruction of SVC leading to
Congestion of face-neck-U.L $\Rightarrow$ Dilated veins
Present 10% of lung Cancer

* any mass in mediastinum cause SVC syndrome so
all causes of BHL lead to it, but MCC is lung
Cancer

* SVC syndrome is an emergency - Best Tx is
radiation $\Rightarrow$ $\downarrow$ size of tumor $\Rightarrow$ relieve compression.

Apical lung tumor = Pancoast tumor



Pancoast syndrome

effect Brachial Plexus

C5-T₁ "MC C8-T₁"



1 - Pain U.L

2 - weakness of small
ms of hand (LMNL)

3 - wasting ms

Horner syndrome

effect Symp Chain



1 = Partial Ptosis

2 = miosis

3 = anhidrosis

4 = enophthalmos

"

Heterochromia if Congenital
horner "NOT acquired"

Tumor effect Diaphragm

1 = Dysnea

2 = Pain in Shoulder b/c same root "dermatome"

→ irritation of Diaphragm → radiation to Shoulder

Case

Pt smoker + Change pattern of Cough

+ recurrent pneumonia + hemoptysis

on Ex -> Rt upper Limb weakness + pain

with small ms of hand weakness esp

little finger

CXR -> Show mass

all of the following are true except =

1 = most probably Dx is lung Cancer (✓)

2 = This Pt mass most probably leading to mediastinum compression (x) 'Apical effect brachial'

3 = on examination Pt has partial prosis

NOTE

- on Ex -> raised JVP -> one of the DD is SVC Syndrome -> MCC is lung Cancer

- on resp ex -> what do you want to see in eye?
eye lid Prosis due to horner syndrome.

1
mets of Lung Cancer 1st to LoN

Then $\rightarrow$ LBLB Bone - Liver - Brain } + Adrenal gland
CRCR Chemo - radio

- Bone $\rightarrow$ Pain + Pathological fracture "from minor trauma"
- Liver $\rightarrow$ Jaundice + Rt hypochondrial pain
- Brain $\rightarrow$ headache + $\uparrow$ ICP + Projectile vomiting + papilli edema
Confusion + Convulsion
- Adrenal gland $\rightarrow$ Adrenal insufficiency

Non-mets manifestation "Para-neoplastic syndrome"

① Endocrinal "tumor secrete hormones"

A • PTH like peptide $\Rightarrow \uparrow \text{Ca}^{+} + \downarrow \text{Phosphate}$

- happens with Sq. cell carcinoma

NOTE $\uparrow \text{Ca}^{+}$ in lung cancer could be due to

PTH "operable" - non met OR due to bone mets "in-operable"

diff by $\Rightarrow \downarrow \text{phosphate}$ with PTH

$\uparrow \text{ALP}$ in bone mets & imaging "scan"

B • ADH $\rightarrow \uparrow \text{volume} + \downarrow \text{Na}^{+}$ "Dilutional" = SIADH

C • ACTH $\rightarrow \uparrow \text{cortisol}$ "Cushing syndrome"

$\hookrightarrow$ So Adrenal gland in lung cancer

($\downarrow$ Hypofunction
by mets)

($\uparrow$ hyperfunction
by ACTH)

D • Erythropoietin $\rightarrow$ B₀M $\rightarrow$ $\uparrow$ RBC "Polycythemia"

So Cancer cause

anemia

"bic $\downarrow$ nutrition-iron"

Polycythemia $\rightarrow$ DD renal cell carcinoma

• rare $\Rightarrow$ Acromegaly due to $\uparrow$ growth hormone

Hypoglycemia "unknown cause"

Thyrotoxicosis

NOTE

"All happen with oat cell "small cell" except PTH
with Sq. cell carcinoma"

So MCC type of lung cancer that cause paraneoplastic
syndrome is small cell "oat cell" "endocrine"

② = Neurological paraneoplastic syndrome "less common"

1 - Cerebellar degeneration "Autoimmune = Ab from Cancer"

$\hookrightarrow$ motor Ataxia

(reversible if Cancer curable)

2 Lambert - Eaton syndrome ?

Ab against Ca^{2+} receptor "pre-synaptic" in nerve

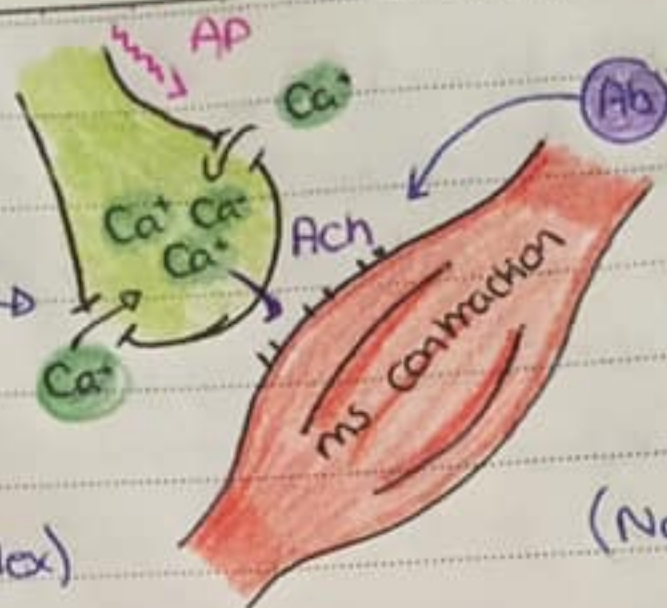
Subject:

Lambert Eaton

Pre-synaptic

↓
Improve after
exertion

LMNL (↓ tone - ↓ reflex)



Myasthenia Gravis

Post synaptic
receptor

↓ After exertion

(Normal Tone + reflex)

3 = Peripheral neuropathy

③ Skin manifestation

- Erythema gyratum repens
- Acanthosis nigricans → DM - malignancy - Cushing
- Dermatomyositis
- Superficial thrombophlebitis ⇒ migratory "Dilated tortuous veins with red skin over it"

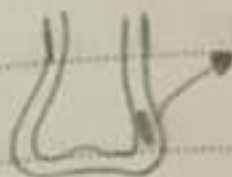
In malignancy
- lung
- Gastro
- pancreas

④ Kidney

nephrotic syndrome

⑤ Hypertrophic Pulmonary osteoarthropathy HPOA

Stage = Clubbing stage IV + Bone pain in radius or ulna
"Drum stick"



due to osteoblastic activity of Cancer in distal end
→ stretch periosteum → pain in wrist or ankle

* mainly with Sq cell & Adenocarcinoma *

C/P Sign

- 1 = normal = so doesn't exclude
- 2 = Collapse = mass completely obstruct airway
- 3 = Consolidation = recurrent pneumonia
- 4 = Pleural effusion
- 5 = Cavitary lesion

General "wt - belt / Appearance / conscious / decubitus / vital sign

Sign of Cushing "moon face - central Obesity"

nasal Polyps

sign of anemia

Plethoric face "Polycythemia

Cyanosis

Prosis

Hand $\Rightarrow$ Astringus - tar stained fingers from cigarette

INU for Dx

- 1) CXR $\rightarrow$ mass - cavitary lesion - pleural effusion
 - collapse - pneumonia - eroded ribs "due to invasion"
 - or fracture

malignancy sign in radiology:

mass in chest > 8 cm more going with malignancy

Shape - Border irregularity - velocity of growth "double in yr"

- No Calcification $\rightarrow$ b/c Ca^{+} usually begin

So for ex: mass 4cm - round - regular smooth surface

No need for biopsy

→ Follow up after 6m if Pt Smoker

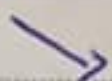
& after 1yr if Non smoker with NO RF

2) HRCT ⇒ if x-ray difficult to show any finding

3) Lung Biopsy ⇒ Confirm Dx

central mass

peripheral mass



Transbronchial

BAL - sputum
specimen

Percutaneous needle

by fiberoptic

show malignant

biopsy under

bronchoscopy

cell fallen in

U/S guide

& take biopsy

airway



or

for Pt that can't

by Transbronchial

take biopsy ex: Old age

U/S → visualize

♥ problem - etc

& take biopsy

"less accurate"

"very accurate"

NOTE

if mass very difficult to take biopsy → take it by
open lung surgery

INV for Complication

CBC $\rightarrow$ Anemia - Polycythemia

Leukocytosis "if pneumonia"

PFT $\rightarrow$ • pre-operative to know Pt operable or NOT

$FEV_1 = < 0.8L \Rightarrow$ In-operable

b/c you will remove part of lung $\Rightarrow$ will $\downarrow$ more

• After Surgery $\Rightarrow$ to see result

ABG $\rightarrow$ RF Type 1 mainly

could be type 2 (if on top of Ch. bronchitis)

• $Na^+ \downarrow$ • ADH $\uparrow$ • $Ca^{+} \uparrow$

INV for mets



LFT

CT Abdomen



$Ca^{+} \uparrow$ - ALP $\uparrow$

Dexa-scan "Dual energy x-ray Absorptiometry"
to see bone density

b/c all cancer cause osteolytic action $\rightarrow \downarrow$ bone density except prostatic carcinoma that has osteosclerotic action $\rightarrow \uparrow$ Bone density.

- CNS $\rightarrow$ CT - MRI
- Adrenal gland $\rightarrow$ CT - scan

level of Cortisol

Synacthen test to know adrenal insuff

PET scan "Positron emission tomography"

for any cancer to know mets

b/c malignant cells take sugar more due to rapid rate of division

so inj pt with (Fluoro-deoxy glucose) "Glucose 18"

isotope sugar $\rightarrow$ image it $\rightarrow$ any hot spot indicate cancer mets

NOTE

Pleural effusion of Cancer

1 = Large amount	] malignant pleural effusion	$\rightarrow$ take sample to cytology to see malignant cell
2 = Bloody "Hge"		
3 = recurrent		

Tx

"Operable" 75% survival rate upto 1yr + 55% upto 5yrs

Pt Not C/I for surgery

1 = Any paraneoplastic syndrome

2 = Pericarditis without effusion

3 = rib - Diaphragm - Chest wall involvement

4 = Trachea involvement 2cm above carina

5 = Pleurisy without malignant pleural effusion

NOT operable '3m survival rate'

1 = T₄ mediastinum invasion 'Local invasion'

(A) Trachea upto carina.

(B) Oesophagus

(C) ♥ involvement with pericardial effusion

(D) mediastinal Nerve involvement $\Rightarrow$ Phrenic N

recurrent laryngeal N

Symp N

(E) malignant pleural effusion 'large bloody recurrent'

2 = N₃ $\Rightarrow$ Contralateral L₄N enlargement (ex: BHL)

3 = M₁ $\Rightarrow$ mets 'any organ involved' LBLB + Adrenal

4 = FEV₁ < 0.8L

5 = severe comorbidity 'severe ♥ problem - COPD - renal - Liver failure failure'

Prognosis $\Rightarrow$ Poor prognosis

Survival rate: 1yr $\rightarrow$ 20-30% / 5yr $\rightarrow$ 5%

Not operable 'Chemo $\pm$ radio'

Chemo $\rightarrow$ kill malignant cell $\rightarrow$ $\uparrow$ survival rate 3m $\rightarrow$ 1yr

radio $\rightarrow$ for Compression Symp + Pain Symp

"Pneumonia"

- Fatal disease - 6th cause of Death
- MC acute infectious disease lead to death is pneumonia
- MC Chronic infectious disease lead to death is HIV
- 2nd Chronic infectious disease lead to death is TB
- Any Pt with cough + fever must exclude pneumonia
(± Chest pain / Dysnea)
- ⊕ FAHM, b/c acute No sever wt loss.

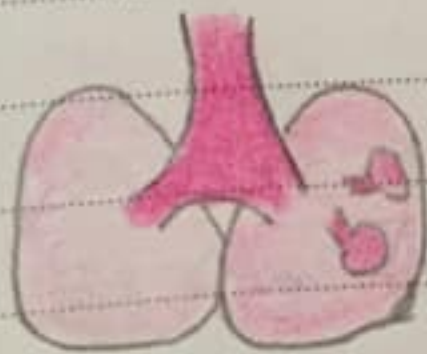
Def acute infection of lung parenchyma "Lower resp"
Characterized by Fever - Cough ± (Chest pain / Dysnea)
⊕ new CXR changes

So normal CXR exclude pneumonia

NOTE

by exam pt may have pharyngitis & tonsillitis by tongue depressor & misdx pneumonia & given amoxicillin & penicillin & its Lower that need parental Tx

So must do CXR b/c pneumonia Fatal!



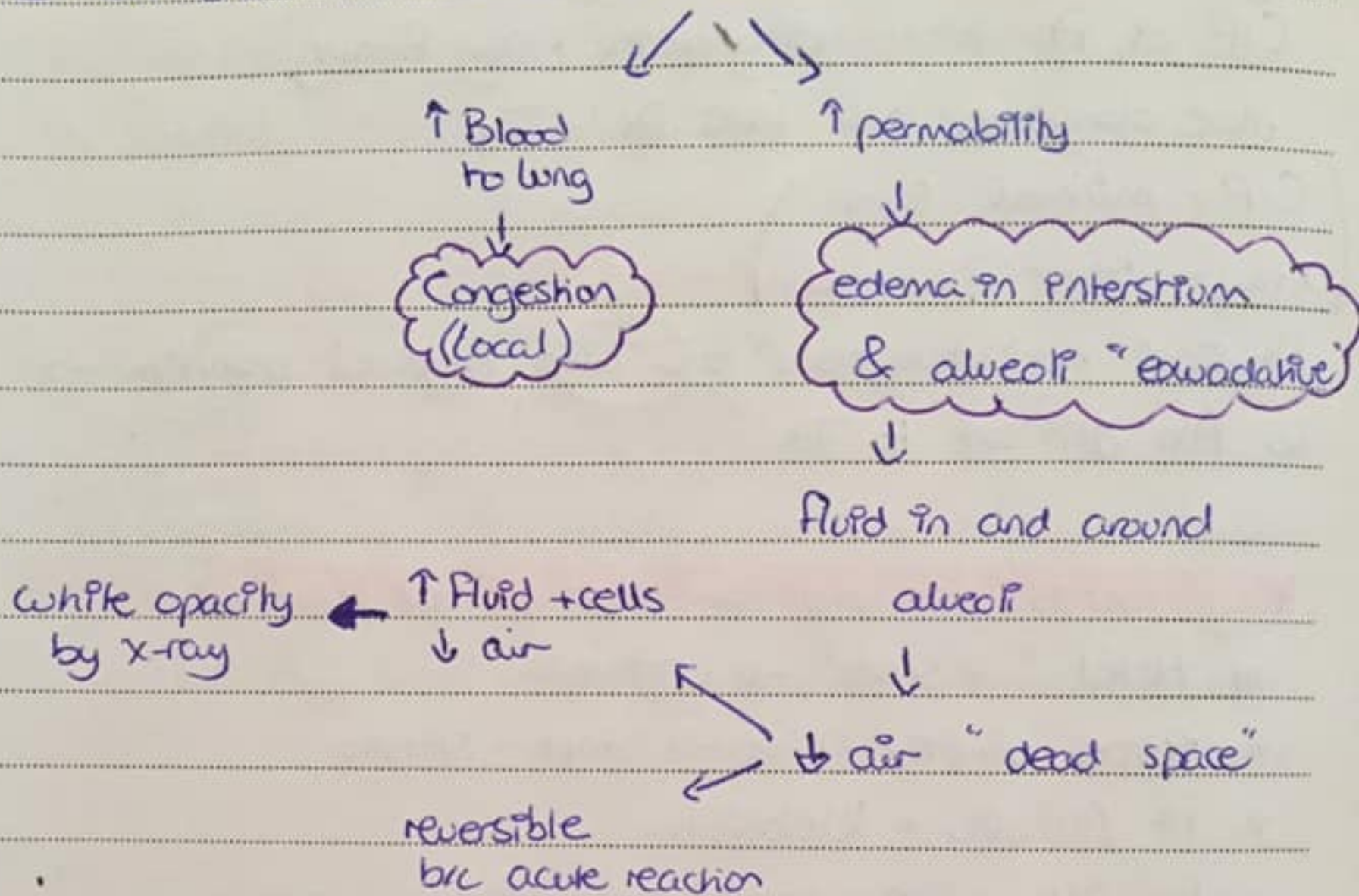
Paranchyma infection → Bact

↓
inflammatory
mediators
enz⁺

← WBC
immune
cells

↑
Chemotaxis
& Cytokines

Inflam chemical mediators $\rightarrow$ V&D



NOTE

Typical

Homogenous opacity
Lobar pneumonia

Uni-lateral

effect one lobe $\leftarrow$ Apical
middle
lower

resp symp mainly
& less constitutional non specific
Symptoms



Atypical

Heterogeneous opacity
Broncho-pneumonia

Bi-lateral "Basilar mainly"

effect Bronchi & interstitial
mainly non-specific symp
"FAHM" & less or No
resp Symptoms

Atypical pneumonia = walking pneumonia
CIP of Pt Not going with x-ray finding
more dangerous b/c Late Dx + Tx

(CIP = minimal symp
x-ray = severe Damage)

Atypical more dangerous b/c also Atypical organism →
so Ab difficult to Tx

Key words for organism that caused pneumonia

- # Hotel "7 stars" → Legionella
- # Herpes Simplex before → Staph - Strepto
- # Pt Alcoholic = Klebsiella
- # Pt DM = Streptococcus pneumoniae
- # Otitis media = H. influenza
- # Hemolytic anemia + Coombs +ve = Mycoplasma
- # Hepatosplenomegaly = Chlamydia

NOTE

Other type of pneumonia ⇒ Interstitial Pneumonia
• mimic ILD

Classification

Community
acquired
CAP

Hospital (HAP)
acquired "nosocomial"
> 48hr of admission

NOTE

medical staff are risk for pneumonia if they stay
in hospital > 72hr "3days" in 90 Days!

Other Classification

recurrent
Pneumonia

↓
any chronic
lung disease
+ ↓ immune

Pneumonia

↓
in
Immunocompromised
Pt (AIDS)

Aspiration
Pneumonia

Prognosis of pneumonia

CRUB₆₅ score ⇒ knows prognosis / Admission or not
type of Tx

C Confusion

R Resp rate > 30

U urea level 7mmol/L

B BP less than 90/60

65 age or more

Score 15

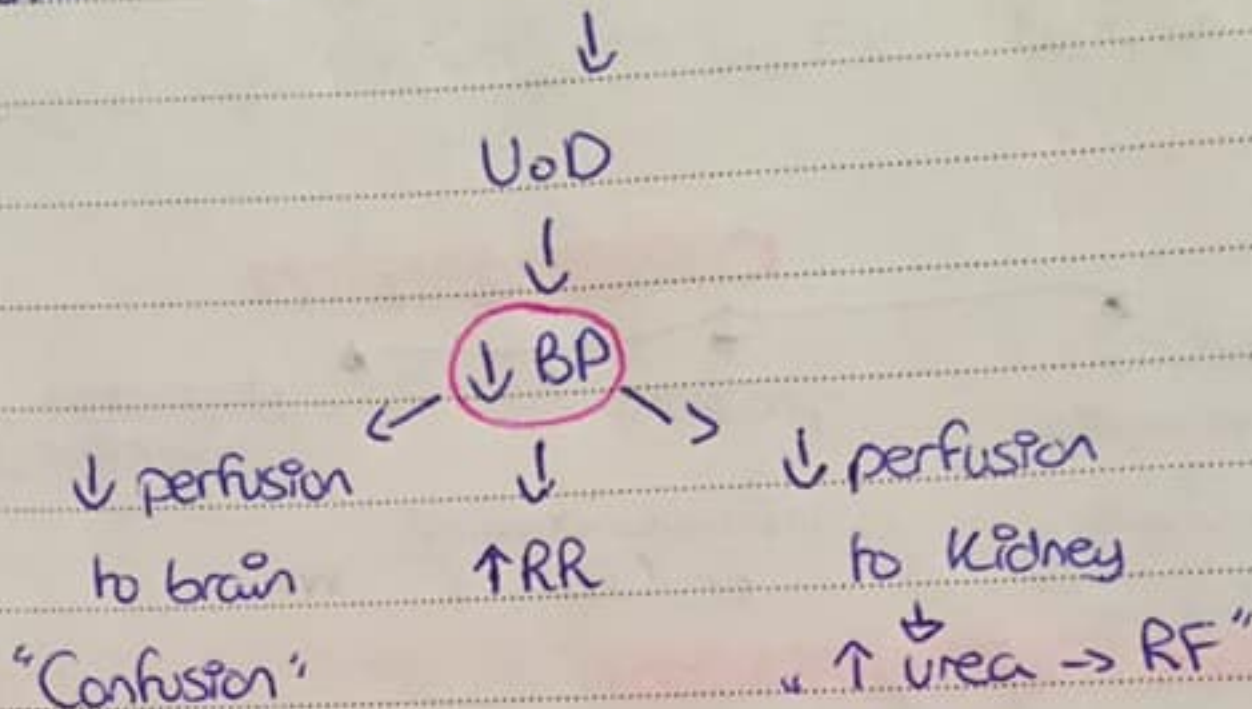
0-1 = Tx at Home + Ab tabs

2 = Admission in ward + Ab IoV

3 or more = Admission in ICU b/c may need MoV

CRUB score b/c

Bact $\rightarrow$ toxin $\rightarrow$ Bacteremia + toxemia + septicemia



NOTES IN X-ray

- PA = V-shaped Clavical
- AP = Horizontal Clavical
- white opacity in normal lung present $\rightarrow$ ribs

Intra-alveolar septum
Palm BoV

Zones of x-ray

- upper $\rightarrow$ First 2 ribs
- middle $\rightarrow$ 2nd to 4th rib
- lower $\rightarrow$ below 4th rib

DD of Cavity

- Cancer Py - 2ny from renal "cannon ball"
- TB
- Pneumonia → lung abscess ex: Staph aureus
Klebsiella

NOTE

Acute disease → thin wall cavity + Abscess still in
air fluid level 

Chronic disease → thick wall cavity + air in it 

NOTE

renal cell carcinoma when mets to lung → in middle
zone → Cannon ball

NOTE

- Fever not always indicate infection

DD ⇒ malignancy - Allergic - Sun stroke - Hyperthyroidism - etc

- Immunocompromised Pt with infection mainly have
No fever or low grade b/c fever from pyrogen
ex: PG that is secreted by immune cells "WBC"
that are not working in immunocompromised Pt :-
HIV - DM - etc

Classification of pneumonia

according
to CXR

- Heterogenous
- Homogenous

according
to C/P

- Typical
- Atypical

according
to infection

- CAP
- HAP
- AIDs
- recurrent
- Aspiration

NOTE = Aspiration pneumonia

Rt side mainly "middle or lower lobe"

→ upper border of Lower Lobe

risk factor

1 = Any pt have difficult in swallowing { ms problem
Nerve problem

ex: CVA effect CNS + 10

Bulbar palsy

neuromus junction disease or ms disease (MG)

Parkinson's disease

2 = Pt under sedation or Drug abuse or Alcoholic

3 = GERD

4 = Achalasia "or any oesophageal motility disorder"

"Poor Prognosis b/c Aspiration of Chemical & bacterial
normal Flora

Risk factor of pneumonia

- 1 = all risk factor of Aspiration pneumonia
- 2 = Children - old age] extreme age $\Rightarrow$ Poor Prognosis
- 3 = Alcoholism - Drug Abuse
- 4 = DM
- 5 = Liver Failure "b/c make Ig" + CKD "prevent loss of Ig by urine"
- 6 = AIDs $\rightarrow$ Poor Prognosis $\leftarrow$
- 7 = Asplenia $\rightarrow$ Autosplenectomy $\rightarrow$ SCA - Eelpac
 $\rightarrow$ Splenectomy
- 8 = Chemotherapy - Steroid "before organ transplant or Tx of Ca"
- 9 = Health care worker "organism in hospital resistant ="

NOTE

Asplenia risk for OPSI "Opportunistic post splenectomy infection"

1 = H. influenza

2 = Influenza viral

3 = Pneumococcal

So HIP vaccine must be given



• if elective surgery $\Rightarrow$ before surgery by 14 days

• if emergency surgery $\Rightarrow$ After surgery by 14 days
"Trauma"

NOTG Case Pt after MoV present with fever + cough
 $\Rightarrow$ MoV as pneumonia
 "MCC organism pneumonias"

Community Acquired pneumonia CAP

CAP Typical

- (FAHM) mild
 $\hookrightarrow$ High grade fever

- Resp Symp = rapid onset & severe

1 = Cough $\rightarrow$ 1st Dry then Productive

MC pneumonia

- Strepto. pneumonia
- H. influenza
- Mycoplasma "Atypical"

note

کرامیل $\rightarrow$ (Brown) rusty in strepto.
 (yellow) PUS "Present in staph"

If severe cough $\rightarrow$ mild hemoptysis "sticks of blood"

- MCC of hemoptysis when organism cause cavity
 ex Staph aureus - Klebsiella

2 = fever

3 = $\pm$ Chest pain

4 = $\pm$ Dysnea

NOTE epigastric pain $\Rightarrow$ DD - Basal pneumonia
- Inf MI "must exclude"
& other GIT 'pancreatitis - gastritis - peptic ulcer - etc'

Atypical pneumonia Constitutional symp with mild
Chest symp

• Mycoplasma • Legionella • Chlamidia • Klebsiella

Sign General

1 = Looks ill - Feverish toxic - distress

2 = Vital sign $\Rightarrow$ • HR $\uparrow$

• BP normal or $\downarrow$ b/c (A) VoD from toxin

(B) anorexia $\downarrow$ fluid intake

(C) D & V

• RR $\uparrow$ (to compensate)

• Temp $\uparrow$

3 = Central cyanosis in tongue in severe cases b/c O_2 sat $\downarrow$

4 = Confusion + disoriented $\Rightarrow$ Bad sign 'Red Flag'

Indicate urea toxin upto brain or hypoperfusion to brain

NOTE

usually pr mainly all sign & symp are free
present only with Confusion

Chest ex 8

1 = Inspection = distress + recession

2 = Palpation = • good Chest expansion except area of pneumonia

••• TVF ↑ at area of pneumonia

• Trachea + Apex beat at place

mediastinum
diverted if
Comp

[except if complicated by Pleural effusion
or pneumothorax => Push
or Collapse => Pull

NOTE Klebsiella is only organism that cause deviation of Trachea & Apex beat

b/c cause gangrenous pneumonia "necrosis + gases"

-> Push (usually apical)

(So Alcohol + Apical pneumonia = Klebsiella)

3 = Percussion => Dull b/c fluid + exudate + ↓ air

if with effusion -> Stony dull

↓
DD • Mesothelioma (Asbestosis)

Cancer of Pleura

NOTE DD Hyper resonant

• Pleural effusion

1 = pneumothorax

2 = emphysema

4 = Auscultation

- ↓ air entry at area of pneumonia
- Type of Breathing = Bronchial *

b/c alveoli is a dead space → so 1st air entry is to bronchi

DD

1 = Trachea

2 = Parasternal "Bifurcation of Trachea"

3 = Interscapula

- Added sounds

Crepitation

early → fine

late → coarse

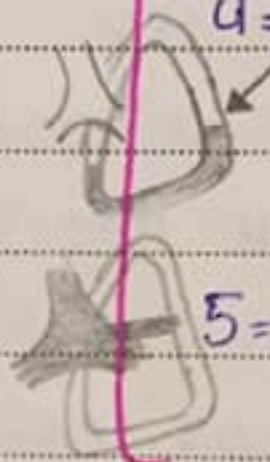
↳ "After resolution & starting Tx"

4 = upper level of pleural

effusion "b/c tissue is pushed by fluid"

5 = Bronchopleural fistula

"Connection b/w airway & Pleura"



• Auscultation Dx pneumonia

- +ve egophony ⇒ tell Pt to say "E" → you will hear it magnified to "AAA" on area of pneumonia
- Whispering ⇒ tell Pt to say "44" → you will hear it magnified

(if Both +ve ⇒ Clinical Confirm pneumonia "Consolidation")

Note 3 Clinically Conformatory tests

GIT $\Rightarrow$ venous hum on Rt hypochondrium $\Rightarrow$ confirm Portal HTN

CNS $\Rightarrow$ Babinski sign + Hypertonia $\Rightarrow$ confirm UMNL

resp $\Rightarrow$ Egophony + whispering +ve $\Rightarrow$ confirm pneumonia

INV

CBC $\Rightarrow$ • leukocyte $\begin{cases} \rightarrow > 20,000 & \text{"sever pneumonia"} \\ \rightarrow < 4,000 & \text{"sever pneumonia"} \end{cases}$

b/c $\downarrow$ immune or due to BM supp from infection

• anemia $\rightarrow$ Coomb's +ve b/c Autoimmune hemolytic anemia in mycoplasma

[* NOTE Coomb's +ve + pneumonia = mycoplasma
Coomb's +ve + leukemia = CLL]

ESR - CRP $\uparrow$

CRP better for monitoring than ESR b/c ESR need time to drop even after Tx

NOTE for follow up & to know improvement

- C/P improvement $\Rightarrow$ $\downarrow$ fever - Cough $\downarrow$ - Chest pain $\downarrow$
- CRP $\downarrow\downarrow$
- NOT CXR b/c opacity remain for 6wks after Tx

So CXR good for Dx but poor inv for follow up

CXR

Opacity of Rt
border of 

⇒ Could be

middle or lower lobe

Diff by Lat view x-ray



Opacity in LF lower border
of  ⇒ indicate lower
Lobe "No middle lobe"

"When you know the lobe
you could suspect the
type of organism"

Microbiological testing

1 = Blood

- Culture & sensitivity → if bacteraemia
- Ag → for Streptococcus
→ for Legionella "By Direct Fluorescence"
(But better in urine)
- Ab → IgM for mycoplasma "cold type" "Coombs +ve"

2 = Sputum or throat swab

- Culture & sensitivity
- gram stain
- Ziehl-nelson stain (AFS) for TB

3 = Urine Legionella Ag "more sensitive"

RBC "hematuria"

NOTE Antibiotic NEVER given before samples
 b/c if given & didn't work $\rightarrow$ can't know
 organism b/c "you changed results" False -ve

Tx O_2 + 3A + Bed rest "good nutrition / IV Fluid"

- O_2 if O_2 sat $< 92\%$ or $PaO_2 < 60$ mmHg
- Antipyretic
- Analgesic } paracetamol
- Antibiotic
 - $\rightarrow$ Blind "empirical" before result of culture
 - $\rightarrow$ Broad spectrum
 - $\rightarrow$ oral or I.V according to CRUB₆₅

oral $\Rightarrow$ Amoxicillin + Clarithromycin "macrolides"

I.V $\Rightarrow$ Clarithromycin + 3rd generation cephalosporines

- " • Cefotaxime
 - Cefixime
 - Ceftriaxone
- } After
sensitivity
test

cr

$\Rightarrow$ Clarithromycin + Augmentin 1g

NOTE

if Pt improved DONT change according to C/S
 & if not improved then change " " "

NOTE if you suspect its strepto $\Rightarrow$ ex: rusty sputum

give Benzylpenicillin "very sensitive"

↓
"B-lactam"

if Pt sensitive
to it

↙
mild skin
reaction

↓

3rd generation
cephalosporines
"β-Lactam"

↘
Anaphylaxis

"Never give B-lactam"

↓

Macrolides

↔ Erythromycin
↔ Clarithromycin
↔ Azithromycin

or

Clindamycin

NOTE Syphilis D.O.C penicillin

if Pt is preg & sensitive to penicillin $\rightarrow$

desensitization of penicillin regimen

Atypical pneumonia

- Mycoplasma $\rightarrow$ Ciprofloxacin or macrolids

+ Doxycycline

- Legionella $\rightarrow$ Ciprofloxacin or macrolids

+ Rifampicin

Pseudomonas $\rightarrow$ "Hospital + M.O.U"

by Antipseudomonas penicillin $\Rightarrow$ Piperacillin - Ticarcillin
or Ciprofloxacin or Ceftazidime.

Staph =>

Flucloxacillin

↓ if resistant

Vancomycin (MRSA)

↓ if resistant

Linezolid (VARSA)

↓ if resistant

call consultant (LARSA) ☹

Atypical Pneumonia

Mycoplasma

FAHM + systemic complication rare

Comp

1 = Bullos myringitis

2 = Erythema nodosum + multiform 'Target lesion'

* 3 = neurological manifestation

- GBS "After pneumonia"
- meningitis - encephalitis
- myelitis "inflamm of spinal cord"

* 4 = Autoimmune hemolytic anemia

"Cold type - Coomb's +ve"

NOTE DD of erythema nodosum

- Sarcoidosis
- post streptococcal
- Idiopathic
- Drug
- TB
- IBD

• Atypical mainly Bronchopneumonia Bi-lat "Heterogenous opacity"

• epidemiology

- more in Autumn
- epidemic every 3-4 yrs
- effect young children → ex: in school

Dx

b/c No cell wall → difficult to Culture so can't have sputum Culture

Dx mainly serology → IgM against mycoplasma
if -ve doesn't exclude ⇒ Do PCR

Tx

Doxycycline + Ciprofloxacin + macrolides

"Penicillin + B-lactam NOT effective "No cell wall"

Legionella

- mainly in summer "7 star hotel" المراجعة
- ⊕ cooling system "key word"

FAHM + systemic manifestation before resp



Broncho or

Lobar pneumonia

Cough + Chest pain



Sever Abdominal

Pain + NVD



Loin pain

hematuria

Prt urea + hypo-

Albuminemia



BOM supp
lymphopenia
WBC < 4000

Sever => Poor Prognosis

10% Fatal

Post Put

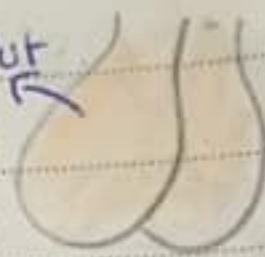
↑ADH

↓DCT

↓ water Absorption only

"dilutional hyponatremia"

SIADH



Dx

by urine → Ag of Legionella
"very sensitive"

IF -ve → serology

Ab in blood

DD SIADH in resp

1 = lung Cancer

2 = Sarcoidosis

3 = Legionella pneumonia

Tx Macrolids + Ciprofloxacin + rifampicin

Key words

- winter = Staph + Strepto
- rusty sputum = Strepto
- herpes labialis = Strepto
- Influenza infection = Staph
- Purulent sputum = Staph
- pneumonia ⊕ pneumothorax
Cavity
emphysema } = Staph

NOTE MC typical => Strepto, MC Atypical => mycoplasma

Klebsiella

Poor Prognosis

- Atypical pneumonia
- gram -ve
- key word $\Rightarrow$ Alcohol - Apical "upper" Lobe - DM
- gangreneous pneumonia $\Rightarrow$ DD Staph

Tx

3rd generation cephalosporine

H. influenza

2nd common cause of CAP

effect mainly COPD Pt $\Rightarrow$

Chlamydia psittaci

- Bird Contact
- risk for EAF

lung fibrosis
pneumonia

- Difficult to culture

Tx = Doxycycline

Organism effect COPD

1 = Strepto. pneumoniae

2 = H. influenza

3 = Moraxella catarrhalis

Organism effect C.F

1 = Pseudomonas

2 = Staph. Aureus

Hospital acquired pneumonia

- risk for health care worker + MoU as pneumonia
- After 48hr of admission

Look for → { new fever
 Purulent Cough
 CXR changes
 ↑ CRP } b/c MC infection
 After Admission
 by 2 days is resp

esp if Pt O_2 requirement ↑ + O_2 sat ↓

Symp start after 2 day & Completed within 5 days

Organism of HAP "Nosocomial"

{ Staph. aureus
 "gram +ve"
 cocci
MRSA } + { 'gram -ve Bacilli'
 pseudomonas
 klebsiella
 E. Coli }

Tx ⇒ I.V 3rd generation cephalosporine → for gram +ve/-ve
 & Gentamicin → for gram -ve

- If you suspect staph → by Purulent sputum / Cavitory etc
 ⇒ give vancomycin

So if Staph CAP → start Flucloxacillin → then vancomycin
 but if HAP → start Vancomycin

- If you suspect pseudomonas → Post MoU
 ⇒ give Antipseudomonas penicillin → Piperacillin
 or Ciprofloxacin
 or 4th generation cephalosporine
 Ticarcillin

Aspiration pneumonia & lung Abscesses

Oro-pharyngeal content Aspirated through trachea mainly to Rt-lung $\rightarrow$ lower lobe upper part or middle lobe
Complicated by lung Abscess

Organism

mainly anaerobic - oral normal flora:

- Staph aureus
- Klebsiella
- Legionella
- Fusobacterium
- "fusiform" } normal flora

+ Chemical substance $\Rightarrow$ eg: GERD $\rightarrow$ HCL



all causing necrotizing pneumonia + gangrene



Lead to cavity + Abscess

NOTE

- HAP more severe than CAP b/c organism is virulence & the Pt is ill "in Hospital"

- Aspiration pneumonia more severe b/c mixed bacteria & Pt is weak "Comorbidity"

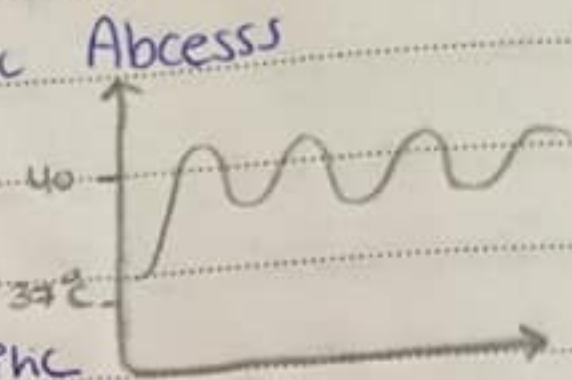
C/P same as CAP

① Halitosis "Bad breath" b/c Abscess

① Fever "Heckic fever"

Fluctuating but Not back
to normal even with Antipyretic

Indicate Abscess



NOTE => Intermittent fever goes back to normal.

① Clubbing rapidly => indicate Abscess

"So pneumonia + Clubbing = Abscess"

Case

PT CVA 20 yrs back having hemiplegia, present with

Cough - Fever - Chest pain

on Ex -> Clubbing + Bad order

on CXR -> Cavitary lesion with air-fluid level



Aspiration pneumonia

Tx O₂ + BA + Drain of Abscess

if present will cause granulation + persistent fever

"لو آتت بالحبوب والحمى المستمرة"



by fistula -> empyema

* Drainage by wide chest tube b/c viscous PUS

• Ab Broad spectrum β -lactam - Cephalosporine

⊕ Ab for anaerobic - D

Clindamycin

Better
↓
Above Diaphragm

(but could be
used in
resp cases)

metronidazole
"Flagyl"

below Diaphragm

NOTE

Drug abuse in case could be → 1 = I.E "Rt side"

2 = Aspiration pneumonia

3 = HIV "AIDS"

Reccurant pneumonia

RF 8

1) Any Chronic lung Problem

Asthma

COPD

bronchiectasis

2) Any Local mass ⇒ Stasis in mucus

Foreign body

tumor

LoN

3) Any pt low immunity

malignancy "multiple myeloma" - malnutrition - extreme age

CKD - Liver failure - HIV - Chemotherapy

Pneumonia in immunocompromised Pt

normally CD4 T-lymphocyte "Cellular immunity"



kill

stim
CD8

"humoral immunity"



stim B-cell



Ig

Count normal
> 500,000

HIV → CD4 ↓ but still > 500,000

AIDS → < 500,000 ⇒ Opportunistic infection

< 200,000

PCP

pneumocystis
carinii
= jiroveci

< 100,000
> 50,000

Toxoplasmosis

< 50,000

MAC

mycobacterium avium
complex



< 50,000 ↑ risk of

CNS lymphoma +

Demyelination

NOTE

when Pt CD4 is 210,000

he is given a prophylaxis Tx

for PCP

Co-trimoxazole "Trimethoprim + Sulphamethoxazole"

3 days / wkly

but if therapeutic → Higher dose - IV - daily

Dx Presentation Atypical "very minimal symp"

Pt Could Be free from symp "So Not exclude

• Pneumothorax could be only presentation

CXR → 10% normal

90% perihilar infiltration "heterogeneous opacity"
"Not specific"

all pneumonia if CXR is normal exclude

except pneumonia in immunocompromised!

HRCT → if normal Not exclude

sputum → Can't be done b/c Dry cough

BAL → stain by Silver Stain or Giemsa Stain

Inv of Choice if imaging normal

but if BAL Normal Not exclude

Lung Biopsy ⇒ GOLD standard

NOTE Exercise induced desaturation

Pt mainly Hypoxic $O_2 \downarrow < 92\%$

at rest For ex: O_2 sat 90%

After exercise sever rapid drop in O_2 sat

"Characteristic for PCP"

Subject:

Tx Co-trimoxazole parental + O₂ + IV fluid
+ Steroid $\rightarrow$ $\downarrow$ Fibrosis + inflammatory process
"So its only pneumonia that tx by Systemic Steroid"

Hydatid cyst

"echinococcus granulosus"

Pt effected by contaminated food with eggs from
dog & sheep feces

egg will rupture and release cysts ~~that~~ that effect
all the body except "hair + nail" $\geq$

Liver & Lung are common sites

if cyst rupture cause $\Rightarrow$ Anaphylaxis

Tx Albendazole

Cyst $\Rightarrow$ thin wall with clear fluid + Calcification

"Never take Biopsy"

$\rightarrow$ "water-lilly
appearance"

Tuberculosis (TB)

- Caused by *Mycobacterium tuberculosis* "Oldest organism on earth"
- 2nd most common chronic infection that is fatal
"1st = HIV / Acute = pneumonia"

- multi-systemic infection characterised by caseating granuloma, effect all body except (Cartilage)

- *Mycobacterium tuberculosis* complex include =>

1 = *M. tuberculosis* "most common organism"

2 = *M. bovis* "Cattle"

3 = *M. africanum*

Lives intra-cellular mainly, Delay growth "Chronic" -> Culture needs 4-6 wk to grow.

Route of transmission

- Air Drop "Cough-sneeze-talking" -> most common
So 1st effected (Pty) is resp system
rare military TB without lung effected
- Ingestion of unpasteurized milk "esp local cheese"
effect GIT + also lung b/c inhalation of TB that is stuck upper airway

MTB

M-bovis

- Skin Contact if Pt has skin lesion of TB & other Pt has wound "open skin" "less common"
- Vertical Transmission: ♀ to Child "less common"

NOTE

(NOT by Blood transfusion)

Prn Organs of TB

Lung

GIT

L.N

Skin

extra-pulm

Hx of Dx of TB

- 1882 → Tuberculin test "PPD"
- 1890 → Acid fast stain "Ziehl-nelson stain" → Bacilli
- 1970 → CXR
- New → Quantiferon test

↳ Counting of Interferon gamma

Note TB is contagious if lung is effected by Cough, but Not if GIT is effected b/c its Not "Feco-orally" Transmitted.

Cylindrical

Red Color

aerobic

Delay growth

in Pulm TB there are 2 types

- Open TB = Infectious Pt
- Closed TB = Non Infectious Pt

Open Pulm TB

Infectious Pt

By BAL & sputum

even 1 +ve sample

over 3 days

(isolated)

& start Anti-TB

After 2wk →
Closed

Closed Pulm TB

Non infectious Pt

By BAL & sputum

3 -ve sputum samples

over 3 days = No Bac

BCG vaccine

- ↓ incidence of miliary TB
- Prevent CNS involvement
- Not from TB

NOTE

MCC of Death in TB Pt is Adrenal insuff when supra-renal gland is effected due to severe hypotension (↓BP)

b/c ↓ Steroid suddenly

So when given Rifampicin "Anti-TB" you must exclude

Adrenal involvement or give steroid before Anti-TB

b/c rifampicin is an enzyme inducer

Epidemiology

Age effected 15 - 75 yrs "Peak = Productive" age

Golden age = 15 - 18 ⇒ Difficult to cause disease

< 5 yrs - > 75 ⇒ severe & spread early in body

↑ low social economic + over crowded "ex: Prison"

TB exposure

- Contact with open TB → TB in lung
"middle + lower lobe"
- 1/3 of world population
- Asymp → pathogenesis Not started
- MQ + neutrophil kill back without help of T-lymph "CD4" → TST -ve
- Immune system Not exposed → CXR normal
- (70 - 90% stay at this stage)
"Good Immunity"

10-30%

b/c ↓ immune or
Large amount of
back

TB infection

- Back get in alveoli → intracellular (MQ)
- Chemotactic for CD4 T-lymph
- secret Interferon gamma, with MQ + epithelial giant cell
- all around back → necrosis & caseation with fibroblast activity → fibrosis → forming "Gohn's focus"

Immune Containing TB

- Limitation Not killing back
- Pt Asymp - CXR usually normal
- b/c minimal reaction + small granuloma "Not Clear"
- TST +ve b/c T-lymph exposed
- (Majority of cases 80% stay Dominant / Latent)

10%

↓ immune

re-exposure
"re-infection"
↑ back

- CKD • Old age
- malignancy • malnutrition
- Drugs = Steroid
Infliximab &
cytotoxic Drugs

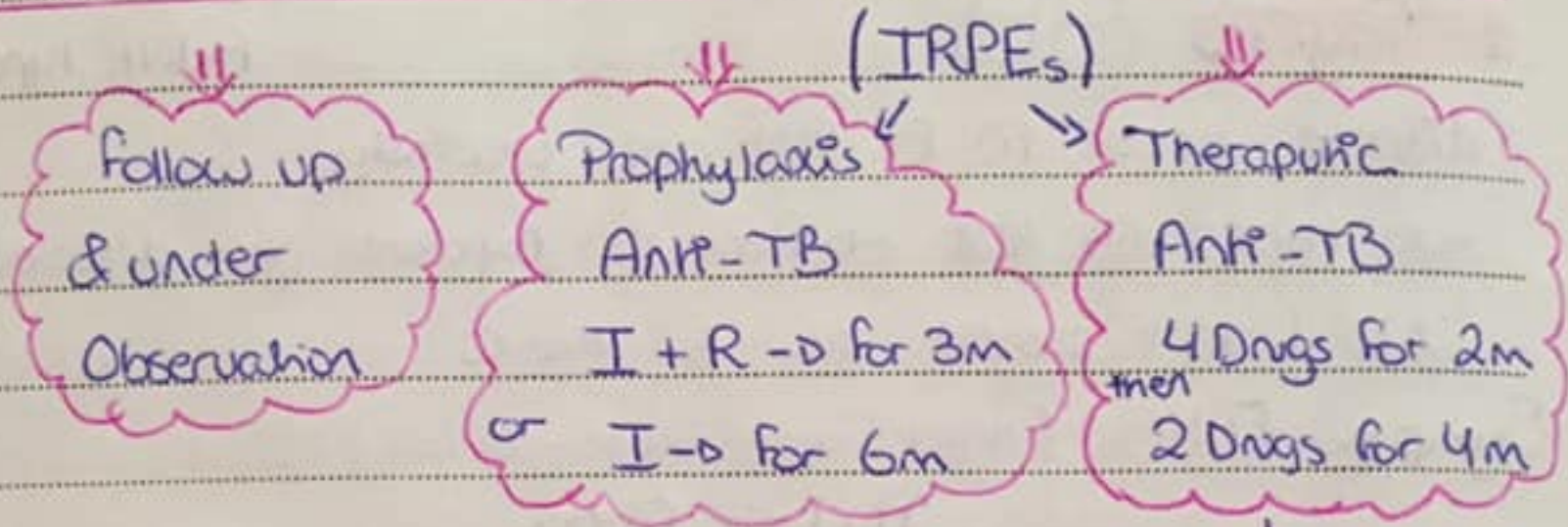
TB disease

Symptomatic effect

Pulm & extra Pulm

- Pulm → Gohn's Complex of Ranke
(cavitary lesion of Gohn's focus + mediastinum L.N enlargement)
- if ↓ immune → millet seeds
- extra pulm → Systemic manifestation
- early IF in 1st 2yrs of exposure
- Late IF > 2yrs of exposure

	TB exposure	TB infection	TB disease
CXR	-ve	-ve	+ve or normal
C/P	-ve	-ve	+ve
TST	-ve	+ve	+ve



NOTE

- Diseased Pt either localized in lung or disseminated to the body according to Pt immunity
- 90% stay in lung "Pulmonary"
- 10% go "extra-Pulmonary"

Total 6m Tx
but if CNS involvement
1yr Tx or more
b/c permanent
Damage & fatal

- Pt with AIDS if exposed to TB will be diseased & 50% present with extra-pulm "disseminated"
- MCC of Death in AIDS Pt is TB
- more contagious "infectious" if Pt AIDS

Presentation

Prim TB

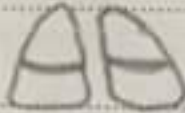
"Child type"

Post Prim TB

= reactivation of Latent

= 2nd TB "re-exposure"

Adult type

I = Prim TB 

disease occur in Pt that are previously

-ve tuberculin test → from 1st exposure got diseased

"No Latent period b/c ↓ immune"

Symp & FAHM + (night sweat - fever - wt loss)

Prim Gohn's Complex

sever LoN enlargement

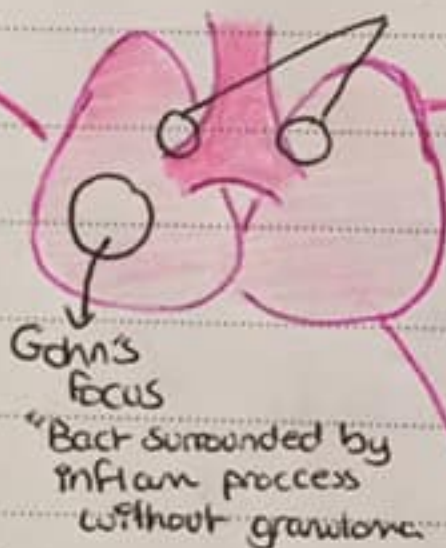
"Lymphadenopathy / lymphadenitis"

TB pneumonia

- Fever → High grade
↳ low if ↓ immune
- Cough / Dysnea / Chest pain
- refractory pneumonia
Not improved by Tx

⊕

Pleural effusion
"para pneumonia" exudative
↑ Dysnea



Compression Symp
"granula phase"

Complete Obstruction of trachea or Bronchi
↓
Collapse

Partial Obstruction
↓
Bronchiectasis

⊕ emphysema

rare → cavitation or fibrosis
b/c ↓ immune

ASS with Erythema nodosum + Conjunctivitis

Miliary TB

spread in lung then extra-pulm

"Child type ⇒ extra pulm > pulm"

So Dysnea in TB due to
1 = Pneumonia
2 = Pleural effusion
3 = anemia of Chronic illness

- Pt w/ TB infection is difficult to Dx b/c 1) short period
- B/c Pt is Asymp what will make you do TST?

If a family member is a known case of TB so family Hx its imp to ask about TB

- If Pt with Any TB presented with pneumonia & Ab was given but Not improved "refractory" $\rightarrow$ misdiagnose Pt will end up in disease state

So Any Pt with Pyrexia of unknown origin or recurrent infection or wt loss & Not responding to Tx with Failure to thrive unexplained $\rightarrow$ must screen for TB by TST "x-ray maybe normal or show Complication"

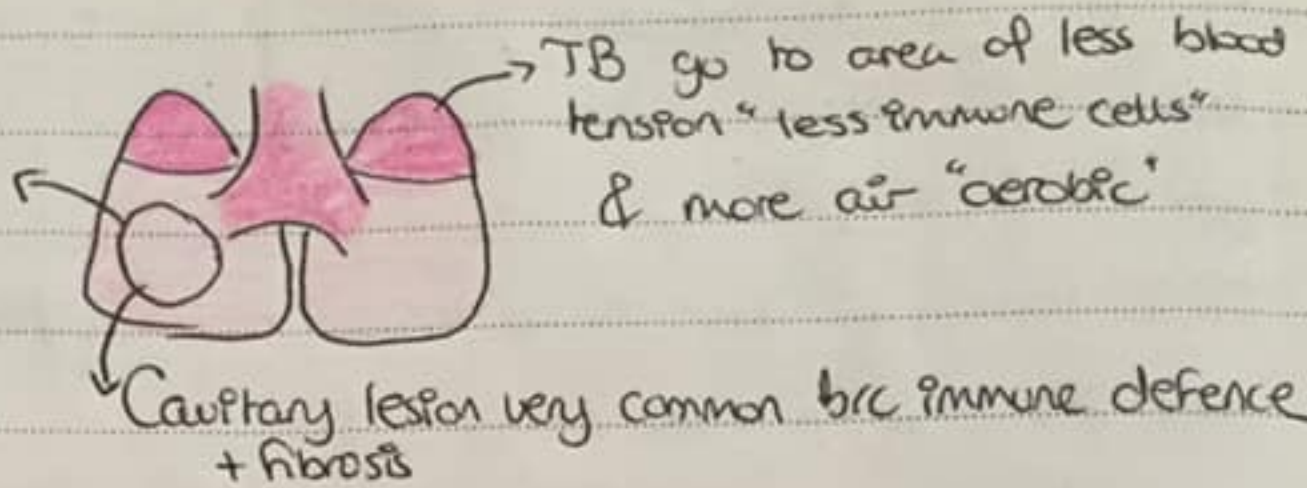
• Trial Dx :

If a child on 2 courses of Ab for chest infection & Not improved $\rightarrow$ give Anti-TB "Isoniazid + Rifampicin"
If improved & fever $\downarrow$ within 4wk $\rightarrow$ Pt is TB

2= Post Priy TB

Adult type = Pt exposed then infected "true TST" for yrs then diseased due to reactivation or re-exposure

TB surrounded by granulation tissue in lung "By biopsy"



C/P

FAHM - (Sever wt loss + night sweat => DD lymphoma)

- Cough -> early Dry / late productive if superposed by infection
- Hemoptysis -> b/c erosion of BV by cavity "Could be massive"
 -> So Prg TB rarely present with hemoptysis b/c (No cavity)
- Chest pain -> indicate pleural invasion
- Dysnea
 "Less military than Prg"

HCR All of the following are ☐ regarding Post Prg TB except:

- 1 = more common in children (x)
- 2 = Commonly ass with lymphadenitis (x)
- 3 = Commonly ass with gohn's focus complex (x)
- 4 = Commonly ass with fibrosis + Cavitary lesion (✓)
- 5 = Commonly in Lower zone (x) Apical

Disseminated TB

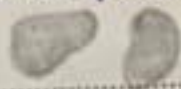
- military = all over the lung field
- extra-pulm = Systemic

Classical

< 40yrs

1 = Reticuloendothelial system RES

[Lymphadenopathy
hepatosplenomegaly]



2 = Serous sac → Pleurisy "Pleural effusion"

↓

Pericarditis "pericardial effusion"

↓

Cardiac tamponade

& Constrictive pericarditis → Diastolic HF

3 = eye → Retinitis

↓

Chorioiditis "Choroidal TB"
= Pathognomic

4 = CNS ⇒ meningitis → Dx by CSF, if -ve ⇒ PCR

NOTE

- miliary TB → ↓ immune → ↓ CD4 T-lymph

So TST falsely -ve

- CXR could be normal b/c ↓ immune

"No fibrosis or Cavity"

So Biopsy to Dx → from Liver in Classical
(show caseating granuloma) from BoM in Cryptic

+ Culture b/c microscopic not always show
granuloma.

Cryptic "Idiopathic"

Atypical

> 60yrs

1 = hepatosplenomegaly

due to extra-medullary

hematopoiesis



2 = Pancytopenia



due to BoM invasion

"CNS - LoN - eye"
NOT involved

- MC TB in hospital = Post Agy
- TB effect all organs except Cartilage

1 = **LoN** $\Rightarrow$ Lymphadenopathy $\rightarrow$ Cervical "1st & MC"

$\downarrow$

- early enlarged red painless & scattered

Axillary

Pelvic

Inguinal

Popliteal

Para-aortic

(No Hotness or tenderness)

- Late matted
- Filled with yellow cheesy material "Caseating" = (yellow white) Cold Abscess
- Soft in consistency [Hard = malignancy
firm = Natural enlarged LN]

Can open into skin by fistula

Note

Cervical normal palpable upto 1cm / patho if $> 1\text{cm}$

Inguinal normal upto 2cm / Patho if $> 2\text{cm}$

- enlarged Cervical called (Scrofula) means Scarf
- L.N become Painful if 1) Pt start Tx
2) Pt take Alcohol

2 = **Skin** $\Rightarrow$ Lupus vulgaris "jelly like purple skin lesion"

3 = **CNS** $\Rightarrow$ most Dangerous b/c sign of meningitis usually absent in TB

Cause granulation tissue $\rightarrow$ meninges stick together $\rightarrow$ CSF can't drain $\rightarrow$ Obstructive hydrocephaly

- Hydrocephaly permanent b/c Fibrosis btw Arachnoid + Pia matter
- Granuloma $\rightarrow$ pressure on Nerves $\rightarrow$ CN Palsy
- must Dx & Tx early b/c Fatal - perinat weakness + paralysis

GIT

- Granuloma in iliocecal joint causing mass in Rt iliac fossa
 $\rightarrow$ Diarrhea & Constipation
- Peritonitis $\rightarrow$ Ascites

DD • Chron's disease
 "Non-caseating granuloma"
 • Transplanted kidney

Bone

- most effect vertebra "Pott's disease" } b/c Blood supply - }
- mainly thorax & lumbar } irregularity of bone - }
- causing loss of normal curve "Gibbs" } immune defence diff }
-  $\rightarrow$ wedge shape due to body necrosis } from other bones }
-  vertebra overlap on each other
- + involve disk $\rightarrow$ Diff b/w Cancer mets (Not involve disc)
- compression on spinal cord
- $\downarrow$
paraplegia
- Back pain + Stiffness

Genitourinary $\rightarrow$ σ epididymitis $\rightarrow$ sterility & infertility
 ♀ PID $\rightarrow$ Subfertility
 kidney $\Rightarrow$ Sterile pyuria

INV CBC anemia of Chronic illness

ESR $>100 \rightarrow$ DD for High ESR ≥ 100

if normal exclude diseased TB
"Active"
used for monitoring also

- 1) TB
- 2) multiple myeloma
- 3) Giant cell arthritis

CRP $\uparrow$

CXR $\Rightarrow$ any finding $\geq$

• pneumothorax

• Collapse

• Cavity

• Pleural effusion

• Bronchiectasis

• mass - fibrosis

or even Normal ==

Bacteriological test "Confirmatory"

- 3 samples in 3 days $\rightarrow$ by AFS $\rightarrow$ even 1 +ve = open TB

if Dry Cough by BAL

test +ve if bact count (50,000 - 100,000)

if less -ve = Closed not infectious

- Other stain can be used = Auramine rhodamine stain

C/S +ve when bact num 10-100 "very sensitive"

so any biopsy send for C/S b/c microscopic could be -ve

Solid media

- Lowenstein Jensen media
- middlebrook media
- "Takes 8wk"

Liquid media

- BACTEC "Best"
- C^{14} radioactivity dependent
- takes 1-2wk
- max 4wk

+ve sputum AFS or Culture $\rightarrow$ Confirm active TB

Tuberculin skin test TST = Mantoux test

Screening pt suspected to be exposed to TB

by PPD from *M. bovis* wall $\rightarrow$ inj in ant surface of forearm in skin $\rightarrow$ wait for 3 days for reaction

by induration Not Diameter

+ve if $\rightarrow$ $> 15\text{mm}$ in Low risk Pt "No Contact with TB Pt"

$> 10\text{mm}$ in High risk Pt "medical staff"

$> 5\text{mm}$ in HIV/AIDS - immunocompromised pt

b/c T-lymp $\downarrow$ so reaction $\downarrow$

NOTE if CD4 $< 200 \Rightarrow$ False -ve, so > 200 for induration

if +ve + Pt Asymp $\rightarrow$ take sputum to know if he

is open "isolation" or Closed TB

False +ve

- BCG vaccine so screen by CXR " $> 15\text{mm}$ to be +ve"

- Atypical mycobacteria "Non-tuberculous"

- Nocardia bacteria

False -ve

- measles - mumps

Defect T-lymp

- Sarcoidosis

- Lymphoma

- Children - Old age "15%"

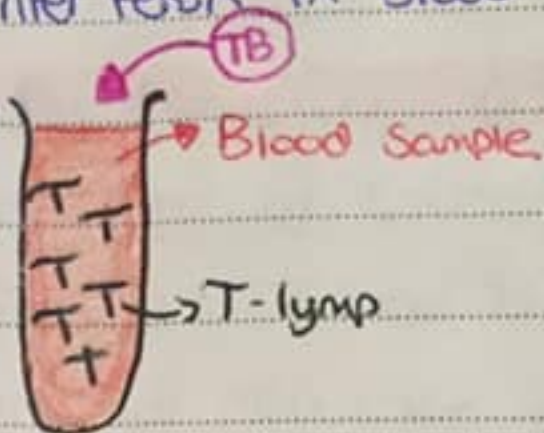
- Drugs = Cyclosporine "Azathioprine"

Steroids

* Pt False -ve $\rightarrow$ How can you know that??
(by Quantiferon test)

Quantiferon = count interferon in blood

if Pt TB $\rightarrow$ CD4 T-lymph
will release high count
of interferon gamma
when exposed to TB
"Counted by ELISA"



if Pt is normal
& never exposed
to TB before
will not release
interferon gamma
"1st exposure"



(give prophylaxis)

Tx Anti-TB HRPEs

[4 Drugs for 2m]
[2 Drugs for 4m]
(6m)

No CNS
or
AIDS

[4 Drugs for 2m]
[2 Drugs for 10m]
(1yr)

CNS / AIDS
↓ immune

HRPEs $\rightarrow$ newist

↓
enz inducer

LFT

exclude Adrenal involvement

b/c all cause

to prevent Crisis or give steroid

hepatitis except (E)

most commonly (R)

* Ethambutol maybe Alternated by (S) b/c resistance to (E) ↑

MOA $\Rightarrow$ All are bactericidal

E + H $\Rightarrow$ effect cell wall "Destroy & prevent building"

R $\Rightarrow$ DNA transcriptase inh

S $\Rightarrow$ Pt synthesis inh

P $\Rightarrow$ unknown

all given orally except (S) IM

S/E

H → vit B₆ Def "So given with it"

Peripheral neuropathy

seizure upto epilepsy

Induce lupus

R → Change color of secretion to ^{red}orange esp urine, tear

RBC hemolysis, ↓ PHT

P → induce lupus

↑ uric acid → gout

S → vestibular neuritis "8th N" ototoxicity

nephrotoxicity

E → Optic neuritis "retro bulbar"

peripheral neuropathy - nephrotoxicity

After Tx of Pt with open TB → see sputum - dif -ve "Closed"

Pt can be discharged and indicate success of Tx
but if remain +ve indicate resistance

Indication for Steroid

- 1 = Adrenal involvement
- 2 = Pleurisy & effusion → ↑ rate of resorption of fluid
- 3 = Pericarditis → prevent constrictive HF
- 4 = meningitis → prevent Obstructive hydrocephaly
- 5 = Anti-TB hypersensitivity

← fibrosis

if A infection state $\rightarrow$ Mantoux +ve or Quantiferon +ve
give ^{2ny} prophylaxis (H $\rightarrow$ 6m or H+R $\rightarrow$ 3m)

BCG vaccine

- on LF arm b/c near to lymphatic drainage "Best area skin immune system of International for statistic studies"
- Prevent only extra-pulm (CNS esp) & military TB

RPO gene study for Bact

if gene +ve in bact indicate resistance to Anti-TB HRPEs
& determine virulence of bact

Types of resistance

- R MTB resistant M.TB $\rightarrow$ to one type of Anti-TB only
- MR MTB multi-resistant M.TB $\rightarrow$ to all 1st generation & some 2nd
- ER MTB Extreme resistant M.TB $\rightarrow$ to 2nd generation $\rightarrow$ need 3rd

DVT & PE

- 1% of mortality rate in hospital
- MCC of Death Post operative "not most common like infection-etc"
- missed Dx

Case Pt with fracture - bed riding for long period present with sudden onset severe dyspnea or Pain $\Rightarrow$ PE

80% of PE are from DVT of LL "Femoral MC vein - then popliteal then Iliac"

PE could be amniotic - fat - air $\rightarrow$ So Not always thrombus

Thrombus cause blood congestion "can't drain up" → so go to superficial system → Dilated tender veins → according to site &

If thrombus in femoral → Dilated vein in thigh

iliac → Dilated vein in all L.L

popliteal → Dilated vein in calf

C/P MC presenting symp "Calf pain"

b/c irritation of congested in blood → inflam response & calf is 1st site of congestion

Pt may present with Pain ONLY without swelling or redness

If thrombus get bigger & de-attach while Pt moving → emboli → IVC → ♥ → Pulm artery → (P/L)

3 Possibilities &

1 = fatal type "most cases"



emboli big enough to close pulm trunk → No blood to lung → No UR to LF side "Circulatory collapse" → Sudden Death

If present early "Pt at ICU for other problem → give thrombolytic by central line "rare"

2 = massive PE



= Sudden dysnea + Hypotension

Close of one major branch → ↓ 50% of blood to lung "Oligemic lung"

→ Pulm HTN + Rt HF + Hypertrophy acutely, ↓ UR → ↓ BP

→ ↓ cerebral perfusion → Syncope

No Pain b/c Pt unconscious from infarction

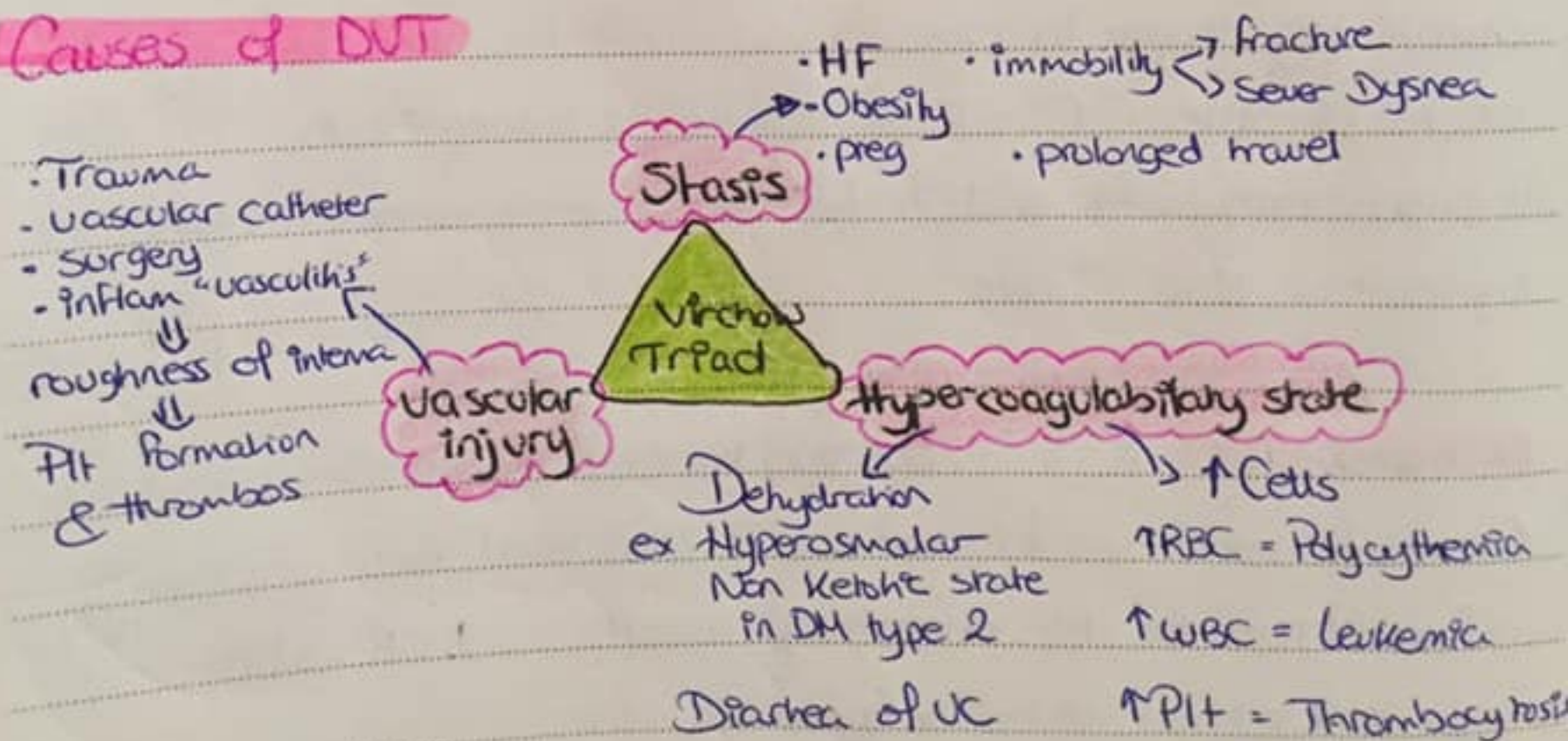
3 = **Pulm Infarction**  -> small BoV occlusion -> necrosis of peripheral lung "wedge shape", b/c good UoR No ↓ BP

- Pain due to necrosis -> inflam mediators to pleura & pleural effusion -> Chest pain "Pleuritic" = any stabbing chest pain related to respiration "DO = Pericarditis - Pleurisy"
- Pain Could be central "if infarction is due to Closure of BoV to medial aspect of lung -> Pleuritic pain at mediastinum area OR Peripheral if branch occluded was lateral"
- Occlusion of small BoV -> Congestion of other BoV + Pulm HTN -> rupture of BoV
 ↳ into Airway "Hemoptysis (10%)"
 ↳ into Pleura "Hemorrhagic pleural effusion"

NOTE

P.E High mortality rate (early) "1st 48hr", Less after that
 "لو ما ماتش بكري من صغوة جيت في"

Causes of DVT



Congenital

- $\downarrow$ Anti thrombin III $\rightarrow$ Deficiency
- $\downarrow$ Ptt C + S $\rightarrow$ Deficiency "ex: SLE / Antiphospholipid Syndrome"
- $\downarrow$ Factor Leiden (5) $\rightarrow$ mutation "Mc hereditary cause of thrombosis"
"or by Liver Disease = $\downarrow$ AT"
- $\uparrow$ Homocysteine $\rightarrow$ Vit B₉ + B₁₂ Def / G6PD
- OCP "esp smoker female >35yrs b/c Lipid disturbance + $\downarrow$ protection of estrogen"
"HDL not working well"

Hx of DVTs

imp to Ask about Drug Hx - bed riding - Travel long time -

Previous thrombosis

Ex of DVTs

- unilat swelling "Asymmetrical" by tape measure >1cm diff
 - Homans sign $\Rightarrow$ dorsiflexion of ankle $\rightarrow$ pain & resistance in calf "Not done anymore b/c risk of detachment"
 - Any Pt suspect DVT $\rightarrow$ Bed rest $\downarrow$ risk of Detachment
 $\rightarrow$ give time for Fibrosis which make it stable
- So Bed rest with Fully catheter or even napkin - I.V Fluid with Anti-Coagulant "heparin - warfarin" $\rightarrow$ prevent the the thrombus from getting bigger $\rightarrow$ being old & stable.
- elevate leg if sever edema / feet stocks late!
 - After 4 days can move slightly - to 7 day = Complete Fixation of thrombus $\rightarrow$ After Full heparinization & INR ≥ 2

DD of sudden onset Dysnea

Any acute $\heartsuit$ or resp problem "PE - MI - HF - Acute angina - tension pneumothorax - Acute severe Asthma - Acute COPD - etc

How to diff b/w real disease or psychological "Panic attack"

by Pulse oxymeter, if $O_2 \text{ Sat} < 92\%$ "organic"

if $O_2 > 92\%$ upto 98% Psychological

C/P Symp $\Rightarrow$ Sudden severe peracute Dysnea

Cough $\pm$ hemoptysis 10% / Chest pain "Pulm infection"

Syncope - Death

Sign $\Rightarrow$ Chest ex: usually normal b/c acute

General ex: Cyanosis "Bad sign b/c $O_2 \text{ sat} \downarrow$ "

$\uparrow \text{HR} / \uparrow \text{RR} / \downarrow \text{BP}$ "Bad sign" $\rightarrow$ massive reactive fever "Stress"

Dx

High risk

V/Q mismatch

1st initial inv

-ve
"Not exclude"

C.T angiography

-ve

Pulm angiography
(Gold Standard)

+ve

Low risk

D-Dimer

1st initial inv

High

-ve
exclude P.E

INV of choice

-ve exclude

+ve
Tx as
PE

+ve

NOTE High risk $\rightarrow$ Old age / Obese / Fracture / Hx of thrombus
Smoker / +ve family Hx / malignancy - etc

Low risk $\rightarrow$ young / healthy / no Hx of thrombus / Non smoker

NOTE

Thrombus = DVT with Fibrin thread $\rightarrow$ normally Plasmin

System break $\rightarrow$ Fibrinogen degradation product FDP = D-Dimer

"very sensitive - not specific for P.E. - $\uparrow$ for any thrombus"

NOTE

• V/Q "ventilation perfusion mismatch"

$\hookrightarrow$ radiolabelled gas for lung $\Rightarrow$ ventilation

& I.V radiolabelled in blood $\Rightarrow$ for perfusion

in P.E. $\rightarrow$ No perfusion, only in alveoli Not in BoV

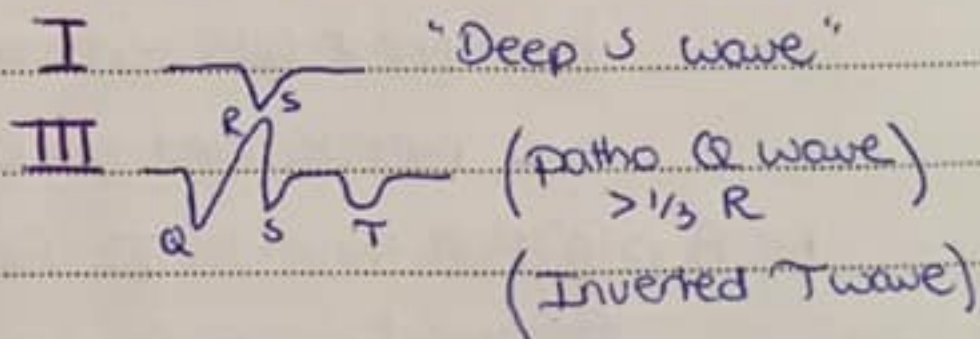
• Pulmonary angiography $\rightarrow$ catheter to Pulm trunk!

INU for Comp

ECG $\rightarrow$ to exclude MI

$\hookrightarrow$ PE Finding = 1 = Sinus tachycardia "MC" sensitive

2 = $S_1 Q_3 T_3$ "10-20%" specific



3 = RVH - RBBB - Rt axis deviation

ABG $\Rightarrow$ RF type 1

Pulse oxymeter $\Rightarrow$ severe hypoxia

CXR $\Rightarrow$ usually normal

- 1 = Oligemic lung "loss of Pulm BoV marking" [Westermarck sign]
- 2 = Proximal BoV in medial aspect congested & Dilated [Palla's sign]
- 3 = Wedge shape infarction [Hampton hump]
- 4 = Pulm effusion "Hge"

INV of DVT

Dopler / Doplex $\rightarrow$ 1st initial simple non-invasive inv

$\rightarrow$ +ve confirm / -ve Not exclude
 $\rightarrow$ show BoV occluded or Not

venography $\rightarrow$ Gold standard "tue confirm / -ve exclude"
show BoV by contrast

D-Dimer $\rightarrow$ Not for Dx / exclude in Low risk Pt

Tx of DVT + PE

Bed rest + good hydration

1 = Anti-Coagulant = Heparin "inj = Iov / S.C" $\rightarrow$ never I.M
"Hematoma"

2 types $\rightarrow$ 1 = **unfractionated heparin**

from mast cell $\rightarrow$ given Iov infusion = work 4-6hr
b/c metabolized by Liver & excreted by kidney

MOA $\Rightarrow$ (A) Anti-Thrombin III Activation "Not work in Cong Def
of Anti-Thrombin 3"

(B) Inhi Factor 9 + 10

S/E $\Rightarrow$ Bleeding - osteoporosis - thrombocytopenia

monitor by = APTT "normal 27 sec"

$\uparrow$ with heparin

2 = **Fractionated heparin** "Low molecular wt" S.C

work 12hr $\Rightarrow$ excreted only by kidney / Not met by liver

So Not given in Pt with renal failure "Longer duration"

MOA $\rightarrow$ inh of Factor 10 only

No need for monitoring "No S/E" - may monitor by activity of Factor 10

"Heparin work immediately - 1st line agent"

(2) **Warfarin** "Orally"

Vit K inactive $\xrightarrow[\text{reductase}]{\text{vit K}}$ active vit K "reduced"
stim $\downarrow$ Factor 10/7/2

(w)

(w) work immediately & inh vit K reductase, but effect after 3 days b/c Pt have already activated vit K

Monitor by PT "normal 13 sec" or "INR normal 1"

After 3 days of given (w)

$\rightarrow$ if INR 2 or more stop heparin & cont (w)

Target = INR 2-3 Not more! For how long?

• if above knee (w) for 6m, if below knee for 3m.

• if recurrent "2nd DVT" for life.

• if with P.E for 1yr at least.

• if Pt with Cong cause of Hypercoagulopathy state $\rightarrow$ for life

NOTE $\Rightarrow$ DVT Cause CVA if Pt have ASD or VSD

Management of PE "emergency" => ABC

- * Any Pt Dyspnoea give O_2 + pulse oxymetre "High Concentration"
- * Analgesia for pain
 - NSAIDs if Pt Not Hypotension
 - Morphine → VOD effect so should be avoided if Pt ↓BP "massive"
- * "So" Vasodilators, nifedipine, Diuretics Not given for BPE b/c ↓BP
- * 2 Large canula → Blood for Inu + IOU Fluid
- * IV Fluid → Crystalloid / Colloid "↑Pt → ↑BP" 500ml
- * Heparin Low molecular wt if suspect DVT
- * measure BP
 - if > 90 = give (w) 5-10mg 1st then according to INR
 - if < 90 = give IVF then dobutamine "↑ve inotropic"
2.5-10 microgram/kg effusion
 - if still ↓ = $\Downarrow$ NE effusion
 - if still ↓ = $\Downarrow$ Thrombolytic IOU

NOTE

IUC Filter prevent emboli from getting up to the lung
in Pt on (w) & have recurrent emboli

Pleural Disease

Normally pleura has no air in it. Slight fluid 10ml max $\Rightarrow$ physiological secretion from arterial & reabsorption from veins & lymphatics

Pneumothorax $\Rightarrow$ Abnormal presence of air inside pleural sac

$\rightarrow$ $\uparrow$ pressure intrapleural $\rightarrow$ lung can't expand $\rightarrow$ **Dyspnea**

Stretch of pleura $\Rightarrow$ **Chest Pain** $\rightarrow$ rapid + severe = emergency

etiology

Traumatic

- Blunt trauma $\rightarrow$ fracture rib & tear skin $\rightarrow$ air in
- Penetrating Trauma

$\Downarrow$
Tx Surgical

b/c also lung & BoV injury

Spontaneous

$\swarrow$
Prim Idiopathic



Young Pt

tall "ex. marfan"

Healthy lung

No Symp

$\searrow$
2ry Diseased lung



> 50yrs

1) emphysema

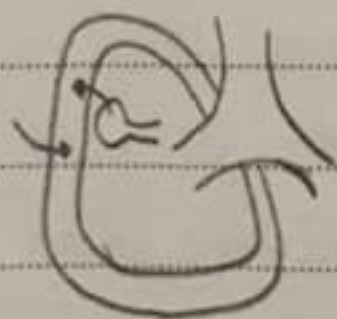
2) Bronchiectasis

3) TB

4) Asthma "rare"

Pathophysiology

1 = **Closed** $\rightarrow$ tear in alveoli or from outside "No continuous



communication b/w alveoli or airway & Pleural sac

$\rightarrow$ small amount of air < 2cm in Diameter $\rightarrow$ then close

simplest type $\rightarrow$ doesn't cause $\uparrow$ Pleural pressure

• Close spontaneously = No Rx

2= **Open pneumothorax** $\Rightarrow$ Fistula btw bronchus & pleura by Chronic Inflamm ex: TB - Bronchiectasis "seen by CT scan"



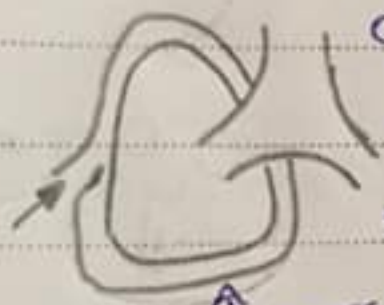
$\rightarrow$ with every inspiration air get in pleura "large amount, but with expiration air get out"

Continuous opening "Pressure variable, intrapleural = Atmospheric pressure, $\uparrow$ & $\downarrow$ with respiration."

Comp $\Rightarrow$ Dust & bact can get in easily b/c Abnormal part doesn't have normal defensive mechanism "cilia + mucos"

$\rightarrow$ risk for emphysema + recurrent inf $\rightarrow$ must close surgically

3= **Tension pneumothorax** $\Rightarrow$ Opening in alveoli make air get in accumulate $\rightarrow$ then close "like one way valve"



can't get out $\rightarrow$ Progressive accumulation of air with every inspiration $\rightarrow$ $\uparrow$ pressure $\rightarrow$ Collapse

$\uparrow$ pressure $\rightarrow$ compression on mediastinal

Heart can't relax "Obstructive Shock" = try to impair & $\downarrow$ vent

Filling due to diastolic failure $\Rightarrow$ DD $\rightarrow$ tamponade

$\rightarrow$ $\uparrow$ JVP, $\downarrow$ BP "No Blood in LF vent", $\uparrow$ HR "Compensatory"

Hypoxic "Cyanosis" b/c collapsed lung

Hyper resonant + silent chest = Lung Collapse

NOTE Cardiac tamponade also cause Diastolic Failure, How to

DIFF ? $\rightarrow$ $\rightarrow$ = JVP $\uparrow$ + BP $\downarrow$ + muffled or Absent $\rightarrow$ sound

($\rightarrow$) = JVP $\uparrow$ + BP $\downarrow$ + No air entry or breathing sound

CIP. Asymp if mild

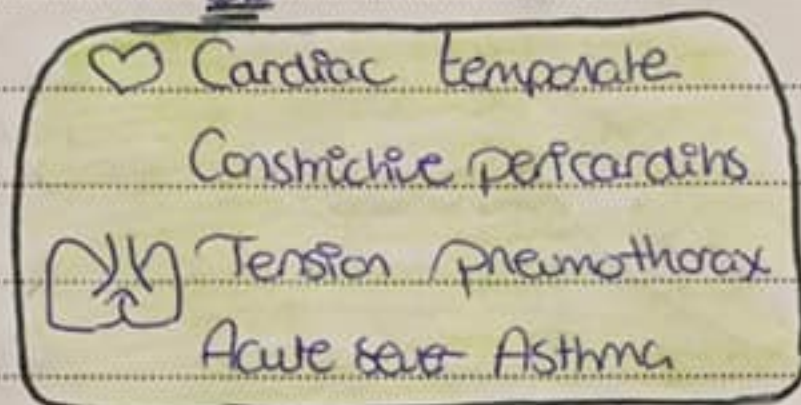
- Chest pain (rapid-sudden-severe) + Dysnea = MC presenting symp
- Surgical emphysema "subcut" → crackles under skin "Traumatic mainly"
if cervical → compression on airway

Sign: JVP ↑ - HRT ↑ - BP ↓ - Pulses paradoxus = ↓ systolic BP

Cyanosis

DD

> 10 mmHg → inspiration



Chest =

multiple -ve sign "↓ expansion - TVF ↓"

Hyperresonant "mostly Apical"

Dx Clinically "Don't Delay management of pneumothorax for
imv"

CXR ⇒ use portable "AP view" if Pt can't stand

- Absent of lung markings - Jet black opacity

if Deviation of mediastinum = Tension

* How to Diff b/w pneumothorax when minimal & emphysema

"air in Pleura or alveoli" ⇒ x-ray can't show so do

U/S or CT-scan

Tx

Any "recurrent 30%"

see Pt Dysnea or rfm > 2cm "Diameter of air in pleura"

Yes

Needle Aspiration
(2nd intercostal space)
mid Clavicular line

Improved

NOT "Failed"

Repeat Needle Aspiration

Improved

NOT "Failed"

Chest tube under water seal

"5th intercostal space
upper border 6th rib b/c VAN
Lower border "prevent trauma"

Bubbling indicate air out → max hrs & stop → Continued indicate fistula

Once stop → Not always good sign

↓
Surgery

• relieve all air → re-inflated "succes" → observation 24hr

• Tube displaced :(

• Tube kinked :(

• Tube obstructed :(

→ air still in → Tx problem

↓
Clamp tube & x-ray

after 2hr

No

Observation 6hr

examination
Pulse oxymetry
x-ray

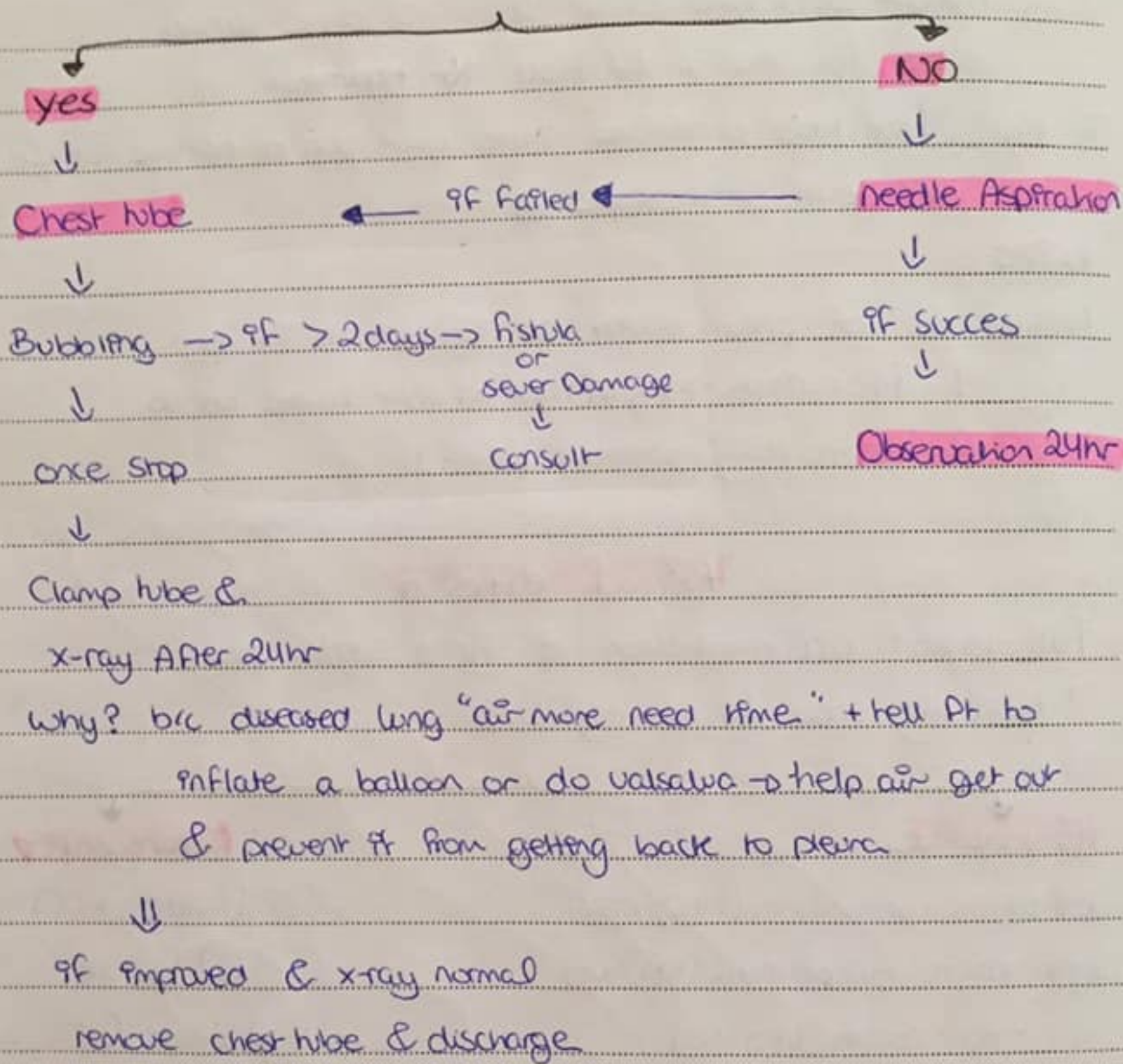
Discharge

NOTE

any Pt on Chest tube
Observation 24hr & repeat x-ray
before discharge b/c risk
for bleeding - Trauma - re-induced air

Subject: Tx 2ry "recurrent rate 50%"

see Pt Dyspnc + 75yr or rim > 2cm



NOTE

Chest tube NOT in 2nd space ? why?

1 = Cosmotec

2 = ms bulk more Ant "Pectoralis" -> so difficult to insert

Tx of ~~pne~~ tension pneumothorax

By Chest tube & before that take vital sign

ABC - ECG b/c Chest Pain - 2 large canula

Blood for inv + IV fluid if hypotension

If No Chest tube available - use your pen to release the air
or your stethoscope $\geq$

NOTE

recurrence of pneumothorax or effusion is Tx by:

or 1 = Pleurodesis $\rightarrow$ inj of material that cause fibrosis

2 = Pleurectomy $\rightarrow$ remove pleura!

Pleural effusion

Pathological accumulation of fluid inside pleura

"Normally max 10 ml by U/S"

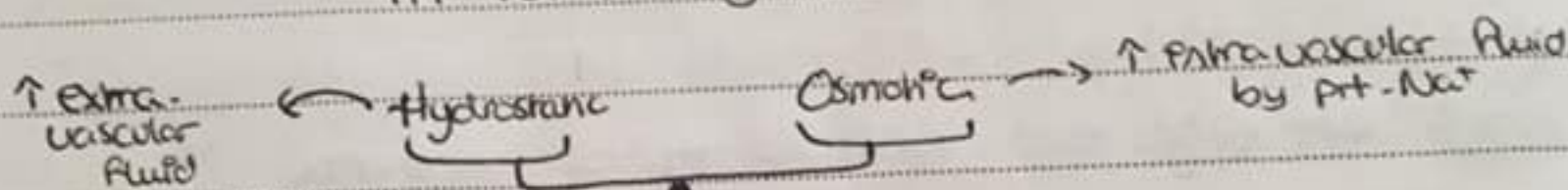
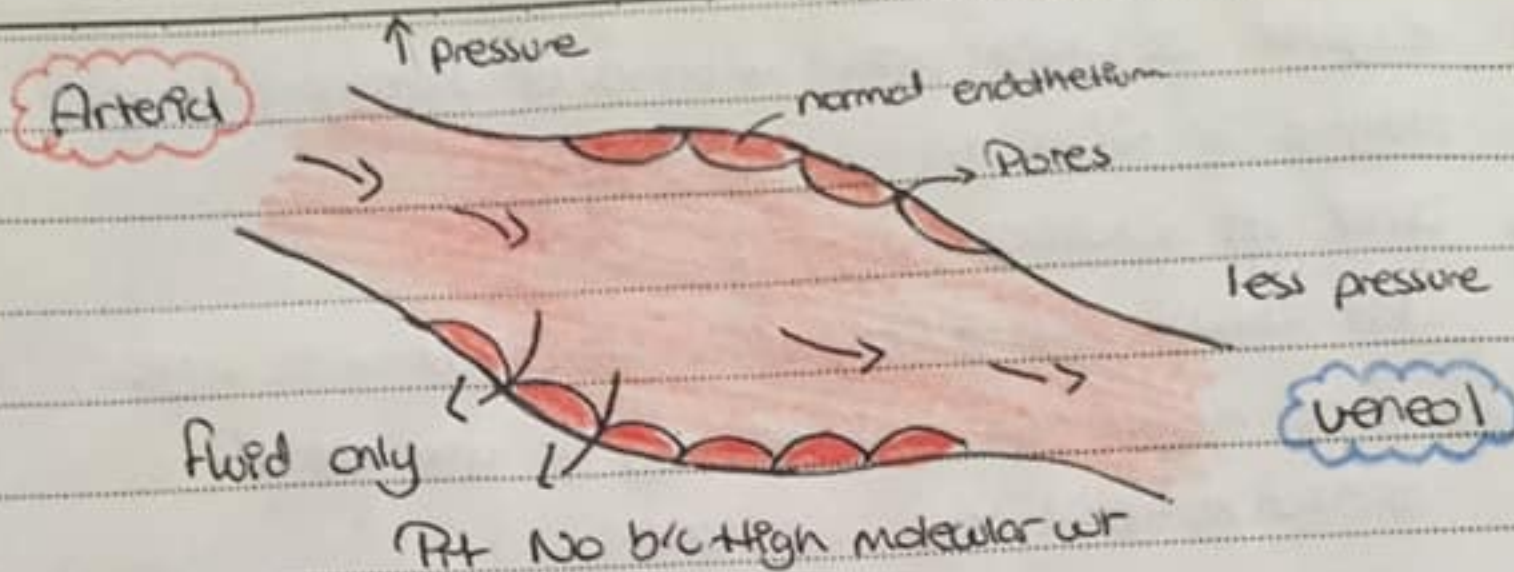
Exudative

- Infection = pneumonia (MCC) / TB
- Inflammation = Autoimmune (RA/SLE)
 - Pancreatitis (L side)
 - hepatitis (R side)
- Infarction = PE 75%
- Trauma "rupture" = esophageal rupture
- malignancy = Breast - lung - lymphoma
- Collapse late

Transudative

- $\heartsuit$ Failure (MCC)
- $\square \rightleftharpoons$ Failure
- Myxedema ($\downarrow$ Thyroid)
- Meigs Syndrome
- PE 25%
(ovarian cyst + ascites + Pl. effusion)
- Collapse early

Subject:



Exudative

Osmotic pressure ↑ extra-vascular
by chemical mediators (PG-IL)
TNFα
→ Pores destruction → Prt out
& water

(> 3gm prt

Specific gravity > 1.018

Prt fluid: serum > 0.5

↑ LDH

NOTE

1st space = BoV (intra-vascular)

2nd space = interstitial → Dehydration from it (interstitial edema)

3rd space = cell → Dehydration from it (cellular edema)

Transudative

Hydrostatic > Osmotic

→ ↑ Hydrostatic → vein

congestion ex: HF

or ↓ Osmotic → ↓ Prt

Cirrhosis - nephrotic

(endothelium & pores normal)

No prt out

C/P Dysnea $\pm$ Chest pain + symp of underlying cause

Asymp PF < 500 ml

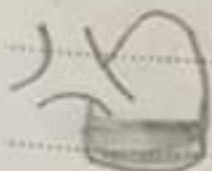
CXR Inv of Choice



Meniscus sign $\rightarrow$ obliteration of costophrenic angle

Fluid higher lat than medially at upper border

Indicate fluid only, ≥ 200 mL



air fluid level indicate hydro-pneumothorax

CXR PF normal NOT exclude (< 200 mL)

U/S more accurate or lateral decubitus x-ray or CT scan

Sign multiple -ve sign + Dullness in percussion or stony

dull PF massive effusion

How to DIFF btw exudative + transudative?

By Dx thoracentesis

1 = Macroscopic examination

Crystal Clear = transudative

Bloody = TB

turbid = exudative

PE

yellow = empyema (AUS)

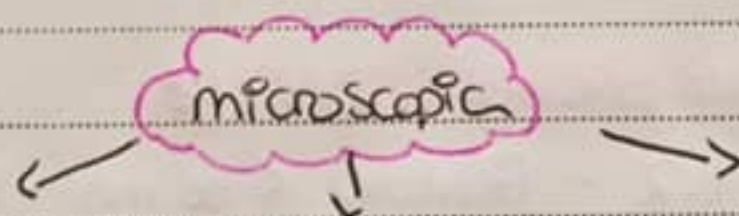
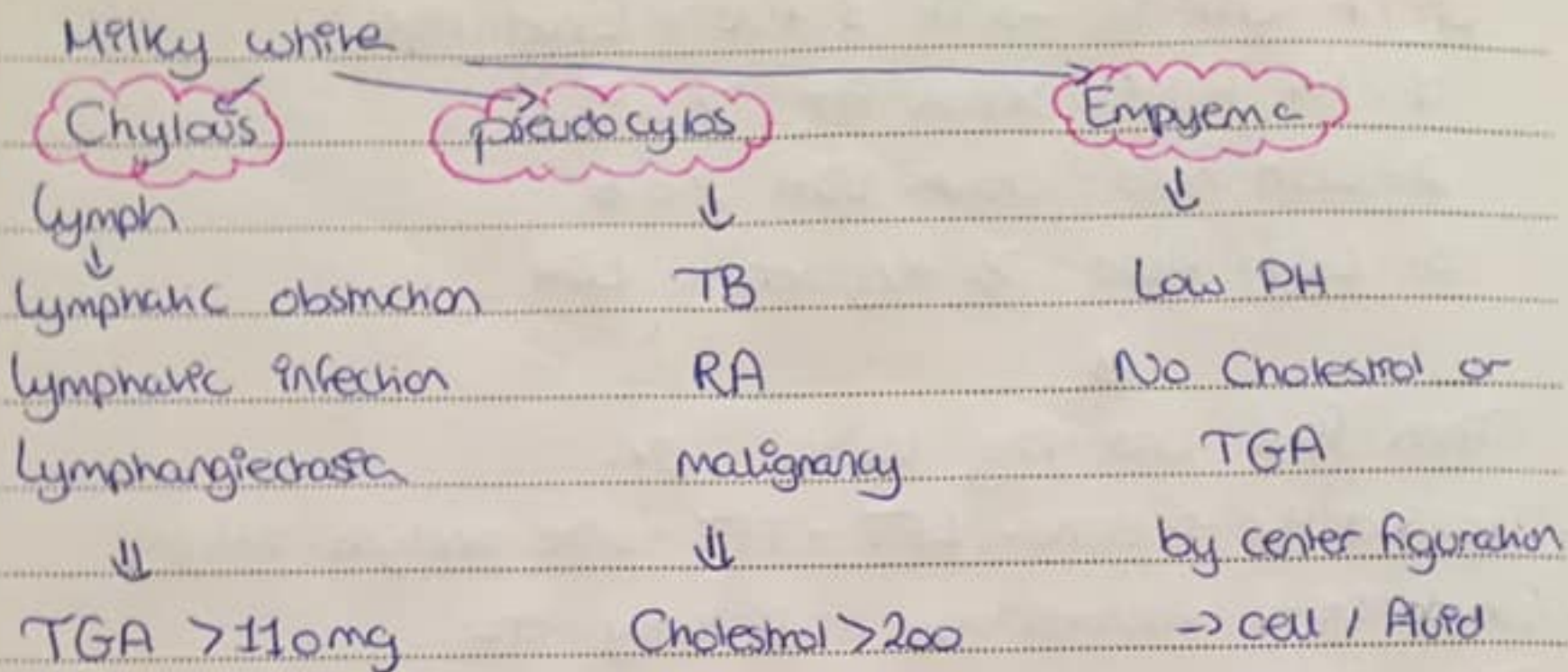
Pancreatitis

yellow green = RA

oesophageal rupture

Bright green = Bact "Strepto-species"

Black = Aspergillus "Fungal"



microbiology

gram stain

AFS

C/S

Cytology

for malignant

cells

Biochemistry

PH

LDH

PH

glucose - Amylase

Interferon gamma } TB

de-aminase

NOTE

Glucose level → very low (RA)

low (infection - malignancy)

PH level → low (infection)

Amylase → ↑ very High pancreatitis "Lipase more specific"

DD ↑ Amylase → Peptic Ulcer - GIT injury - oesophageal rupture
malignancy - hepatitis - etc

Light Criteria $\rightarrow$ if 1 +ve = Exudative

1 = Rt Fluid : Serum Rt > 0.5

2 = LDH Fluid : Serum LDH > 0.6

3 = LDH Fluid $\geq 2/3$ serum LDH



Then find underlying cause ex:

- Transudative $\rightarrow$ echo - LFT - TFT - urine analysis - imaging
- Exudative $\rightarrow$ microbiology - malignancy - etc

Tx TUC

Indication of Parapneumatic Drainage

emphysema - PH < 7.2 - Glucose < 60 mg - Lobulated effusion

gram stain + C/S +ve

NOTE

Therapeutic thoracocentesis to relieve symp not more than

1.5L at one episode bc $\downarrow$ pressure rapidly $\rightarrow$ re-inflation

of lung which will cause vascular bed to expand &

$\downarrow$ pressure intralveolar that will cause Pulm edema

"re-expansion pulmonary edema"

♥ « علم الإنسان ما لم يعلم »